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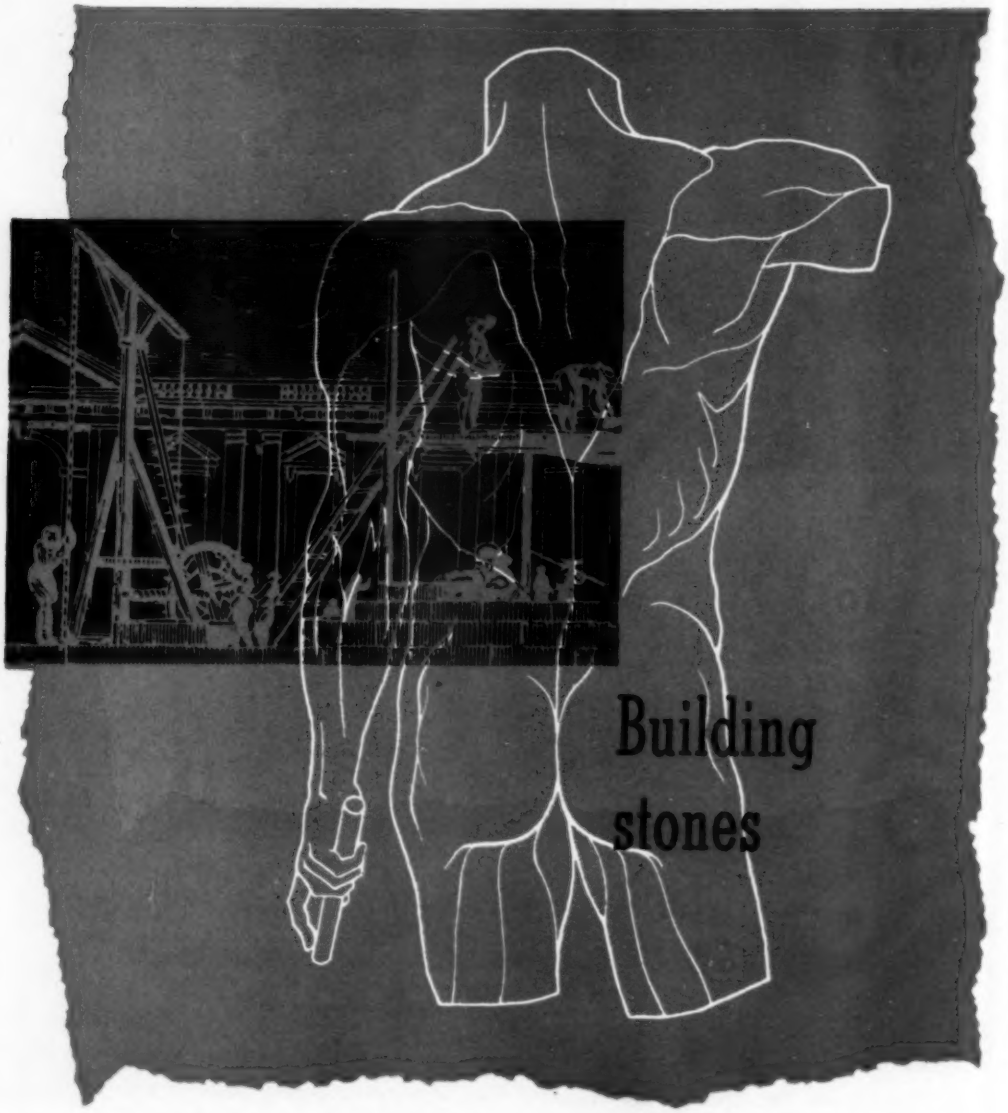

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By the end of July of this year we will have lost almost one-third of the physicians and dentists now serving with our Armed Forces. Without an increased inflow of such personnel, the shortage will assume even more dangerous proportions by December of this year.

These losses are due to normal expiration of terms of service. The professional men who are leaving the Armed Forces during this critical period are doing so because they have fulfilled their duty-obligations and have earned the right to return to civilian practice.

Without sufficient replacements for these losses, we cannot continue to provide adequate medical and dental care for the almost 1,700,000 service men and women who are the backbone of our nation's defense.

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To alleviate this critical, impending shortage of professional manpower in the three services, I am urging all physicians and dentists who were trained under wartime A. S. T. P. and V-12 programs under government auspices or who were deferred in order to complete their training at personal expense, *and who saw no active service,* to volunteer for a two-year tour of active duty, at once!

We have written personally to more than 10,000 of you in the past weeks urging such action. The response to this appeal has not been encouraging, and our Armed Forces move rapidly toward a professional manpower crisis!

Many responses have been negative, but worse—a great number of doctors have not replied. It is urgent that we hear from you immediately!

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Volunteer now for active duty. You are urged to contact the Office of Secretary of Defense by collect wire immediately, signifying your acceptance and date of availability. Your services are badly needed. Will you offer them?

Louis Johnson

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THE TREATMENT OF SINUSITIS

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THE present concepts of the treatment of sinusitis are largely based on three principles: (1) preservation of functioning structures, (2) restoration of normal function and (3) elimination of pathologic abnormality. The success which attends both medical and surgical treatment of infection of the paranasal sinuses is dependent upon balanced achievement of all three principles rather than upon complete achievement of any one with sacrifice of the other principles.

ANATOMY

The lateral wall of the nose is characterized by three overhanging shelves, the inferior, middle and superior turbinate bones, which contribute to the formation of three recesses referred to as the inferior, middle and superior meatuses. The inferior and middle turbinates are well marked and prominent. The superior turbinate is small and is usually obscured from view by the middle turbinate.

The paranasal sinuses arise from invaginations of the nasal mucosa in the middle and superior meatuses. The sinuses open into these spaces throughout life. The sinuses are divided into two groups by the middle turbinate. The anterior group consisting of the frontal, anterior ethmoid and maxillary sinuses arise from the middle meatus and lie anterior and inferior to the middle turbinate. The posterior ethmoid and sphenoid sinuses arise from the superior meatus and are situated superior and posterior to the middle turbinate. The turbinates afford considerable protection to the ostia of the sinuses.

The nasal mucosa is notable for its ciliated epithelium, its mucous glands and its bed of capillaries which are capable of great distention when filled with blood. The sinus mucosa resembles the nasal mucosa from which it arises, but has fewer glands and fewer capillaries than does the nasal mucosa.

PHYSIOLOGY

The defenses against infection which are peculiar to the nose and paranasal sinuses are provided by the ciliated epithelium and its overlying blanket of mucus.[3] The nasal mucus is sufficiently viscous to resist penetration by bacteria, has bacteriostatic properties and is kept in constant motion by action of the cilia. The direction of ciliary action in the sinuses is toward the ostia of the sinuses. This mechanism, when in a state of health, is able to empty the sinuses of secretions. Ciliary action in the nose is directed toward the choanae and is most intense in the region of the middle meatus. Action of the ciliated mucosa and its mucous blanket is important in removing both virulent microorganisms and the products of infection from the nose. Distention of the capillary bed and consequent swelling of the mucosa can block the nose and prevent entrance of irritants.

PATHOLOGY

The pathology of inflammation of the nose is basically similar to that of other tissues of the body. Certain manifestations of inflammation are peculiar to the nose because of the anatomic and histologic characteristics of the nose and its tissues. Obstructed nasal breathing is a prominent symptom owing to the fact that the nasal

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chambers are of limited capacity and bounded by rigid bony walls, and owing to the rich capillary network in the nasal mucosa. The profuse flow of mucopurulent discharge results from the presence of numerous glands in the mucosa. Pain is aggravated by pressure which occurs when tissues swell in a rigid-walled cavity. The symptoms of obstructed breathing and pain occur much more often in acute than in chronic inflammations of the nose.

Repair of acute inflammatory changes in the nose results in the formation of fibrous tissue in the mucosa and, in certain instances, metaplasia of epithelium from ciliated columnar to stratified squamous types. The change resulting from a single episode of acute inflammation is small, but repeated attacks may lead to cumulative damage which is significant in producing chronic inflammation. Inflammation around the blood vessels of the nasal mucosa is particularly significant.[2] Periarteritis results in decreased nutrition of the tissue. Periphlebitis and perilymphangitis restrict the return flow of blood and tissue fluids and cause edema. The end result of these phenomena is tissue necrosis and fibrosis. Thickening of the nasal mucosa due to fibrosis and edema, and loss of ciliated epithelium are important causes of decreased drainage from the sinuses and favor the development of chronic inflammation.

Factors which interfere with the normal defense mechanism of the nose favor the establishment of acute infection. Local obstructions to nasal respiration which slow the flow of the mucous blanket, dryness due to local obstructions or to excessively dry air, loss of ciliated epithelium or temporary paralysis of ciliary action may allow virulent organisms to multiply and become implanted in the nasal mucosa. Chilling the body results in reduced blood flow to the respiratory mucosa. Many infections of the upper part of the respiratory tract begin in the nasopharynx where the epithelium is nonciliated, and spread to the nose. The presence of chronic infection in the pharyngeal lymphoid tissue or the tracheobronchial tree may lead to recurrent episodes of rhinitis and sinusitis.

The development of chronic infection of the paranasal sinuses is dependent upon factors which interfere with drainage and ventilation of the sinuses. Severe acute infections may become chronic if restoration of drainage is not accomplished by therapeutic measures. Obstruction to the ostia of the sinuses favors the development of chronic sinus infection. The ostia of the sinuses may be blocked by deviations of the nasal septum, enlarged or cystic middle turbinates, anomalies of the ethmoid cells, local areas of dryness, nasal polyps, hypertrophy or edema of the mucosa, or metaplasia of the nasal mucosa. Debilitating diseases and reduced general resistance to infection favor the development of chronic sinusitis.

TREATMENT

The treatment of sinusitis is most effective if it is planned to fit the needs of each individual patient. Consideration of the three fundamental principles, namely, preservation of tissues, restoration of function and elimination of pathologic processess, will aid in selecting the plan of treatment best suited to the individual patient. The requirements for treatment of sinusitis vary with the stage of the disease and will, for convenience, be discussed under the headings of acute, subacute and chronic sinusitis.

Acute Sinusitis.—Medical measures fill the dominant role in the treatment of acute sinusitis.

General measures such as rest and support should be applied according to the severity of the infection. Sedative and hypnotic drugs are useful. Control of humidity is important. Dry air leads to drying of the mucous membranes and interferes with the discharge of secretions. The relative humidity should be maintained at 50 per cent or over for best results. Humidifiers which operate without heat result in less condensation of moisture in the room and are preferred to steam kettles. Drugs of the sulfonamide series are useful in controlling infections of the air passages. The value of the sulfonamides is limited in cases of suppurative sinusitis because the drugs are inactivated by para-aminobenzoic acid which is present in pus. Dosage should be adequate to maintain concentrations of 10 to 12 mg. of the drug per 100 cc. of blood, and the patient should be protected against urinary complications by the administration of fluid and alkali sufficient to produce 1,200 cc. of alkaline urine daily.

Antibiotic drugs have largely superseded the sulfonamides in the treatment of suppuration of the sinuses because their activity is maintained in the presence of pus. Penicillin is the most useful antibiotic at the present time. Davison found that 81 per cent of sinus infections were caused by organisms sensitive to peni-

cillin in concentration of 0.2 unit per cubic centimeter. Streptomycin should be used only if bacteriologic study shows the infecting organism to be of the gram-negative group. I prefer to administer antibiotics by intramuscular injection. The infecting organisms in sinusitis lie within the tissues as well as on the surface of the mucosa and are most effectively reached by an agent circulating in the blood. Agents administered by inhalation do not enter the sinuses in significant quantities.

Local medical measures are especially useful when the infection is not severe. Vasoconstricting drugs such as ephedrine may be applied to the nasal mucosa to reduce swelling and promote drainage from the sinuses. Prolonged use of vasoconstricting agents tends to produce secondary congestion of the mucosa which may defeat the purpose for which the drug was applied.

Astringent drugs, an example of which is colloidal silver protein, promote discharge of mucus from the glands of the mucous membrane. Shrinkage of the membrane results. These drugs depress ciliary action. Silver salts may be deposited in the skin, conjunctivas and mucous membranes with resulting discoloration. This complication usually results only after prolonged use of the drug.

Solutions containing therapeutic agents may be displaced into the sinuses by the Proetz and similar methods, or they may be directly instilled into certain sinuses by catheter. Sulfonamides and antibiotics applied in this manner do not remain in contact with the organisms long enough to be of much value. The greatest value of such treatment probably lies in the mechanical removal of pus from the sinuses.

Physical treatments are useful in cases of acute sinusitis. Heat by local applications or diathermy stimulates blood flow to the inflamed tissues and brings comfort to the patient. Roentgen therapy reduces congestion in acutely inflamed sinus mucosa and may bring dramatic relief of pain.

Surgical treatment is generally contraindicated in acute sinusitis because of the risk of osteomyelitis. A noteworthy exception is the trephine operation on the floor of the frontal sinus in cases of fulminating acute infection. Care should be exercised not to disturb the diploic bone of the anterior table of the frontal sinus. Fracture of the middle turbinate toward the septum is a safe procedure which is often useful in cases in which the middle meatus is narrow.

Subacute Sinusitis.—This or the terminal stages of acute sinusitis often benefit from lavage of the affected sinus. This is readily accomplished in the maxillary sinus which can be reached via its natural ostium in the middle meatus or by puncture through the inferior meatus. The approach through the natural ostium is preferred for therapeutic lavage when the ostium is accessible. When the ostium cannot be entered without trauma it is better to make a puncture opening away from the ostium than to damage the mucosa at the ostium. The ostium of the sphenoid can be entered with a cannula in most instances, the nasofrontal duct in a few. Van Alyea has advocated breaking through obstructing ethmoid cells in order to reach the frontal sinus with a cannula. The ethmoid cells can rarely be reached by cannula.

Chronic Sinusitis.—Sinusitis becomes chronic because of some factor which prevents drainage of the sinus by normal physiologic processes and which fails to correct itself spontaneously or as a result of medical treatment. Such factors fall into two classes; namely, anatomic obstruction and pathologic changes. Examples of the former are spurs or deviation of the septum, enlargement or displacement of the turbinates, or anomalies of the anterior ethmoid. Examples of the latter are thickening and fibrosis of the sinus mucosa, nasal polyps, and hypertrophy of mucosa in the middle meatus. Because of these factors the treatment of chronic sinusitis often requires surgical measures in addition to medical treatment.

Lavage of the sinuses is effective in a few cases of chronic infection, particularly when used in conjunction with some procedure for correction of an obstruction to drainage. Procedures for relief of obstruction to the ostia of the sinuses include fracture, crushing or submucous resection of the middle turbinate, submucous resection of the nasal septum, removal of septal spurs or removal of nasal polyps. The submucous resection of an enlarged or cystic middle turbinate allows relief of obstruction to the middle meatus without loss of mucosa.

Cases of chronic sinusitis in which considerable change has occurred in the sinus mucosa often require new drainage pathways for discharge of secretions from the sinuses. Following adequate drainage of pus, the sinus mucosa may

recover to a marked degree. Studies by Sewall and by Snitman indicated that removal of the sinus mucosa is not necessary in most patients operated upon for relief of chronic infection, and that the regenerated mucosa which lines the sinus after removal of its mucosa is less able to resist subsequent infection than is the original lining of the cavity.

Adequate drainage of the maxillary sinus results from removal of a portion of the naso-antral wall in the inferior meatus. The approach through the canine fossa is indicated only in those cases in which the sinus is subdivided by a bony partition, or in which the presence of a foreign body such as a tooth root or a dentigerous cyst is suspected. Drainage of the ethmoid cells is accomplished by exenteration of the ethmoid with removal of the intercellular septa. Exenteration of the anterior ethmoid exposes the ethmoid recess of the frontal sinus, and this procedure is often adequate for drainage of the frontal sinus. Demonstration, by roentgen examination, of dividing septa within the frontal sinus, or orbital extensions of the ethmoid cells may suggest the need for external approach to the frontal or ethmoid sinuses. Osteomyelitis of the frontal bone and infection complicating osteoma of the frontal or ethmoid bone are definite indications for the external approach.

The sinuses comprising the anterior group are more subject to infection than are those of the posterior group. This is due to the protection from the inspiratory air stream afforded the superior meatus by the middle turbinate. The maxillary sinus, due to its dependent location, is more subject to chronic infection than are the frontal or anterior ethmoid sinuses.

The posterior ethmoid and sphenoid sinuses may be approached by one of three routes. The face of the sphenoid sinus may be removed and the posterior ethmoid exenterated by working forward from the sphenoid. The posterior ethmoid may be entered from the anterior ethmoid and the dissection carried into the sphenoid. This is the most direct route when the orbital approach is used. The ethmoid and sphenoid may be reached from the maxillary sinus when the route through the canine fossa is used. The choice of approach depends on the preference of the surgeon.

The rigid walls of the sinuses present special problems in surgical treatment of infection in these cavities. The cavity cannot collapse and obliterate itself as does an abscess of the soft tissues after drainage. The cavity, therefore, remains and is subject to reinfection. The anatomic limitations on the size of the drainage pathway results in all too frequent closure during the healing process. This outcome is particularly troublesome in the frontal sinus. The problem of secondary closure of the surgically enlarged nasofrontal duct has not been satisfactorily solved. Tubes of various materials, mucous membrane and skin grafts, and irradiation by roentgen rays and radium have been used with indifferent success.

The influence of chemotherapy and antibiotic therapy in bringing about resolution of acute sinus infection has resulted in a marked decrease in the incidence of chronic sinus infection. This is truer of the frontal and ethmoid sinuses than it is of the maxillary sinus, probably owing to the insidious course of infection in the latter sinus which does not cause the patient to seek relief in the early stages of infection. The need for surgical treatment of sinusitis is decreasing.

The antibiotics, used alone, seldom result in cure of chronic sinusitis. These drugs are of great value as an adjunct to surgical treatment of the sinuses. The administration of antibiotic drugs during the postoperative period controls infection present in the mucosa and thereby facilitates healing, shortens the period of convalescence and reduces the incidence of complications.

SUMMARY

Treatment of infections of the paranasal sinuses is based on three principles; namely, preservation of functioning tissue, restoration of function and elimination of pathologic processes. The plan of treatment selected for the patient should aim at the solution of his individual problem in the light of these principles. Medical methods are favored in acute infections, but surgical measures are often required for relief of chronic infections.

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HODGKIN'S DISEASE: HISTOLOGIC-CLINICAL CORRELATIONS

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RARELY in the literature on Hodgkin's disease is there a discussion of the significance of lymph node necrosis, fibrosis and invasion of the capsule as to their relationship to the clinical characteristics. The significance of the lymph node eosinophils, neutrophils or Sternberg-Reed cells is seldom mentioned. How much lymph node fibrosis is to be attributed to x-ray therapy, or how much is due to spontaneous sclerosis, usually is not indicated. The tissue slides from 150 cases of Hodgkin's disease have been examined with these and other objectives in mind. The findings in these few cases, although not statistically adequate, are at least provocative.

The data on Hodgkin's disease is frequently presented in the form of age and sex incidence; sites of original lesions; tuberculous and occupational relationships; hematology, survival and treatment information. These interesting correlations, and others, for the 150 cases reported here, will be presented elsewhere.[1]

The material logically fell into the three histologic variants of paragranuloma, granuloma and sarcoma. The term paragranuloma, introduced by Jackson and Parker[2], seems to have been quickly accepted, but does name a type previously recognized and referred to by several other terms, some of which were cumbersome, vague, confusing, or not widely used [atypical Hodgkin's disease (Warthin[3]), Hodgkin's lymphoma (Bersach[4]), "early Hodgkin's disease," etc.]. This paragranuloma type typically reveals a lymph node having an abnormal architecture which, however, may not be completely bizarre, but may retain some follicles and even an occasional identifiable sinus. The main impression is made by the diffuse sheets of mature-appearing lymphocytes that occupy most of the tissue, distorting the normal lymph node pattern. A careful examination will reveal a few isolated typical Sternberg-Reed cells, upon which the specific diagnosis is based. Plasma cells may be seen in moderate numbers, eosinophils may be present, but are scanty, fibrosis is absent or, rarely minimal, and necrosis is lacking.

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When this Hodgkin's disease material was clinically correlated, many of the findings were similar to those noted by other authors. It was not possible, however, to add evidence to support Jackson and Parker's belief that those cases with the paragranuloma variant have a more favorable course than the classical granuloma type. In studying the two variants of Hodgkin's granuloma, the ever-present danger of confusing them with other lymphoma exists. Since paragranuloma and Hodgkin's sarcoma show atypical cytological characteristics, they may be confused histologically with somewhat similar tumors which are not true Hodgkin's disease. This error in proper classification is probably most likely to occur in the Hodgkin's sarcoma variant.

The histologic variations that may occur in the Hodgkin's disease lymph nodes will be considered under variations in lymph node structure or lymph node cytology. The mean survival of the different groups of cases on which microscopic slides were available varied from 13 to 20 months. This is considerably lower than the total mean survival (41 months) for all the Hodgkin's disease cases (with and without available slides) in this group.

Structural Variations in Hodgkin's Disease Lymph Nodes

Necrosis: (Table I) Necrosis may be very marked in Hodgkin's disease, occasionally causing the tissue to be grossly indistinguishable from tuberculosis. It rarely becomes caseous. Necrosis was entirely absent in 74 per cent of the lymph nodes. The remaining one-quarter showed it in various degrees, with four per cent of them having it to a great extent. All of these lymph nodes were examined for tubercle bacilli, and none was found.

There was no significant difference in the incidence of necrosis in those lymph nodes which had been treated with the x-ray (six months or more before biopsy), and those which had not received x-ray therapy.

The cases in which the lymph nodes had little or no necrosis lived on the average much longer (three times or more) than those with moderate or much necrosis.

TABLE I.
Incidence and degree of lymph node necrosis in 121 cases:

Incidence and degree	Number Cases	None	Slight	Moderate	Much
Lymph node necrosis	121	74%	11%	9%	4%
Necrosis correlated with x-ray:					
Lymph nodes without x-ray	58	70	15	8	5
Lymph nodes with x-ray	36	80	11	5	2
Necrosis correlated with survival time in months	35	13±12	18±16	5±7	1±1

TABLE II.
Incidence and degree of lymph node fibrosis in 107 cases:

Incidence and degree	Number Cases	None	Slight	Moderate	Much
Lymph node fibrosis	107	4%	28%	47%	19%
Fibrosis correlated with x-ray:					
Lymph nodes without x-ray	56	5	25	50	19
Lymph nodes with x-ray	37	8	18	49	24
Perivascular fibrosis	100	29	26	30	15
Fibrosis correlated with survival time in months	35	7±2	12±17	16±15	8±9

Fibrosis: (Table II) Selerosis is one of the four primary histological characteristics of classical Hodgkin's granuloma, and yet little has been written as to its natural evolution. The actual evaluation of the cytological effect of x-ray therapy on the nodes of Hodgkin's disease, is difficult. Often the biopsy material is not selected so as to best determine this effect. After x-ray therapy and the resolution of the treated nodes, it is entirely possible that the exuberant new tumor proliferation would not necessarily reflect the direct effect of the previous x-ray therapy. The originally irradiated, small node might be quite sclerotic. Several lymph nodes obtained one to two weeks after x-ray therapy did show an immediate reaction of increased prominence of the reticulum and a decrease in the expected number of cells. Specific material was not available to determine whether these treated nodes could be expected to revert to their former appearance. Certainly, with excessive radiation some fibrosis would be expected.

It is usually stated that the fibrosis increases with the duration of the disease, and such was occasionally the case in this series. However, such was not always so, and just as frequently the final picture presented the same or an increased cellularity over that of the original structure. Increased fibrosis was not necessarily found in the nodes removed from a previously irradiated area. It is very likely that the fibrosis present in enlarging lymph nodes is mostly an inherent characteristic of each particular Hodgkin's disease process. That it probably does not represent a host "resistance," or a relative degree of "differentiation," and hence lessened malignancy, is suggested by the failure of those showing moderate or much fibrosis to survive significantly longer than those with slight or no fibrosis.

A peculiar distribution of the fibrosis was noted in the lymph nodes which were examined. This was in the form of concentric rings of peri-vascular fibrotic tissue, often giving the blood vessels a broad, laminated-appearing adventitia. This was noted in 70 per cent of the lymph nodes.

Capsule invasion: Extension of the disease process across the capsule of the lymph node is usually considered to occur rather late in the disease. Lymphosarcomas, however, are supposed to destroy capsules more easily, and the lymph nodes tend to become matted together earlier. In the lymph nodes of 108 cases, 75 per cent revealed no capsule invasion, and 25 per cent did. This difference in these two groups of lymph nodes was not reflected in any changes in their respective survival rates.

Cytological Variations in Hodgkin's Disease Lymph Nodes.

The Sternberg-Reed cells, the eosinophils and the associated chronic inflammatory granu-

lomatous background constitutes the characteristic Hodgkin's granuloma. As the picture becomes more cellular, especially if of a rather predominant reticulo-endothelial type, with associated mitoses and pleomorphism, the sarcomatous varient is approached. In this series, Hodgkin's sarcoma constituted 11 per cent of the 121 cases. It revealed an average survival of only one-half as much as in the other types of Hodgkin's disease.

Eosinophils: (Table III) These striking cells may occur in such astounding numbers in cases of Hodgkin's disease that the microscopist finds it difficult to believe that they are not of some great significance. What significance they may have has not been established. They were present in various degrees in 96 per cent of the lymph nodes; and in 25 per cent they are in great abundance. It was not possible to relate them to necrosis, fibrosis, nor, indirectly, to the type of fever. Likewise, the number of eosinophils does not influence the subsequent survival of the patient.

Sternberg-Reed Cells: The classical giant cell of Hodgkin's disease is best referred to by the combined name of Sternberg-Reed. Each author has described a particular cell type, either one or both of which may exist in Hodgkin's disease tissues. Bersach[4] suggested that the large cell with abundant cytoplasm and deep staining multiple nuclei described by Sternberg[5] is a later derivative of Reed's[6] "large endothelial cells with vesicular nuclei, hazy chromatin network and one or more nucleoli." A diagnosis of Hodgkin's disease cannot be made microscopically without the presence of these cells. Examination of the table reveals that even in the 17 per cent which had these cells in the greatest abundance, an influence on survival is not apparent. This supports the belief that the Hodgkin's sarcoma variant should be diagnosed not on the basis of greater number of the Sternberg-Reed cells alone, but instead on the existence of other criteria of malignancy—mitoses, cell pleomorphism—especially when existing in sheets of reticulo-endothelial type cells.

Langhans giant cells, in the absence of tuberculosis, are rare in Hodgkin's disease. In this series, one or two such cells were encountered in 6 per cent of the 121 cases.

TABLE III.

Incidence and degree of cellular types:

Cellular type and incidence	Number cases	Percentage	Survival time in months
Eosinophils	123		
None		4	———
Rare		14	10±10
Slight		24	13±13
Moderate		32	15±19
Much		24	12±11
Sternberg-Reed	121		
None		—	———
Rare		2	13±11
Slight		27	14±16
Moderate		51	11±14
Much		17	16±18
Langhans*	121		
None		93	
Rare		6	
Slight		—	
Moderate		—	
Much		—	
Plasma	118		
None		22	
Rare		—	
Slight		37	
Moderate		34	
Much		6	
P-M-N Leukocytes**	116		
None		46	20±14
Rare		—	———
Slight		24	12±14
Moderate		13	9±10
Much		15	1± 1

* in autopsies with no tuberculosis.
** 35 cases only available for calculating survival times.

Polymorphonuclear Neutrophils: (Table III) These calls were absent or insignificant in number in 46 per cent of the 116 cases. They more or less paralleled the presence of necrosis. In 15 per cent they were in great numbers, usually tending to be focal in distribution, and occasionally existing as frank abscesses. As was true with necrosis, they were correlated with survival, so that when present in large numbers they were clearly associated with a decreased survival expectancy.

Miscellaneous calls: Plasma cells are present to some extent in 78 per cent of the nodes, and in 6 per cent they were present in great abundance. No effort to correlate this with survival trends was made. The prominence of lymphocytes in nodes is evident, since one variant of Hodgkin's disease (the paragranuloma) is that type verging toward the pattern of the small cell lymphosarcoma. It was not possible to demonstrate a significantly greater survival time for this type.

Interchange of Hodgkin's Disease and Other Malignant Lymphoma Types

The change of a paragranuloma to a Hodgkin's granuloma or to a Hodgkin's sarcoma is well known. In the Hodgkin's sarcoma stage, the process may closely resemble and merge imperceptibly into a typical example of any of the varieties of reticulo-endothelial sarcoma. For those who believe Hodgkin's disease is one of the typical lymphosarcoma variants, this evolution is expected. Those who favor an inflammatory granuloma etiology, conclude that this only indicated that Hodgkin's disease tends to be a pre malignant granuloma from which a true neoplastic lymphoma may develop. However, when a typical lymphosarcoma develops into a typical Hodgkin's disease process (Herbert, Miller and Erf[7]) the evidence in favor of its being only a variant of the lymphosarcoma group seems to be enhanced.

In this series of cases, the transition of Hodgkin's disease into other malignant lymphomas was rarely observed. Perhaps much of this was due to the infrequency of multiple biopsies. Two of the autopsies were considered as examples of Hodgkin's sarcoma, whereas their previous biopsies were Hodgkin's granuloma. In two instances the original biopsies were characteristic of a giant follicular lymphoma. Subsequent studies revealed one of them to have become a Hodgkin's paragranuloma and the other an active Hodgkin's granuloma.

Lymph Node Viral and Bacteriological Studies

The lymph node tissue was cultured from 24 cases of Hodgkin's disease and from 15 cases of controls (carcinoma, sarcoma, leukemia). Special attention was directed toward the presence of the Brucella organisms. Sometimes bacteria were isolated during the initial inoculation into chicken embryos of the ground lymph nodes. These studies reveal (Table IV) that 12 Hodgkin's disease lymph nodes were without bacteria and 12 contained them. Brucella organisms were not encountered, and the majority of the bacteria seen were of the diphtheroid type.

In recent cases of Hodgkin's disease the lymph node material, cell-free extracts, were passed through multiple series of fertile chicken eggs. An increased egg mortality has been reported by this method,[8] thus lending support to the presence of a possible viral agent.

Autopsy Findings in Hodgkin's Disease Cases

Necropsies were performed on thirty patients with Hodgkin's disease. Only two of these revealed a Hodgkin's sarcoma picture, the remainder showed the typical Hodgkin's granuloma process. The detailed microscopic characteristics of the tumors were not correlated with the clinical picture of the disease, because it was

TABLE IV.

Lymph node cultures:

Cultures	Hodgkin's Disease	Carcinoma, leukemia, lymphosarcoma
Negative	12	8
Staphylococci	0	2
Bacilli	2	4
Mixed staph. & bacilli	2	0
Unspecified organisms	8	1
Total	24	15

felt that the entire process at autopsy was so advanced as to have little or no clinical significance. Instead, a simple tabulation of tumor distribution, other diseases present and organ sizes were considered more informative. (Table V.)

The involvement of the lymph nodes, spleen, liver and lungs was of the expected degree of frequency. The bone marrow tumor incidence in the 20 cases examined was 75 per cent. This figure was obtained by a routine section of, usually, only a single piece of bone marrow from a lumbar vertebral body. A more thorough survey would have doubtlessly increased this percentage of involvement. In fact, it is highly likely that, as Steiner[9] states, the percentage of bone marrow involvement found at autopsy is mostly a function of the effort that the pathologist makes to find it. Unusual organ distributions or bizarre primary sites were not encountered in this series. The liver and spleen weights were obtained. Their mean weights were increased. However, in 68 per cent of the livers and 21 per cent of the spleens the weight was within normal limits.

Particular attention was directed toward the presence of tuberculosis. It was present in five (17%) cases, being active in three of them and inactive in two. In two of those with active tuberculosis it was impossible to positively separate every lesion due to tuberculosis from those of Hodgkin's disease. Similar confusion has been noted in cases of histoplasmosis with Hodgkin's disease (Miller, Keddie, Johnstone and Bostick[10]). However, biopsy or autopsy slides did not show any of the specific infectious granulomas producing a picture absolutely characteristic of a classical Hodgkin's granuloma. It was always "atypical."

Other diseases present at autopsy are listed (Table VI). None was remarkable, or apparently correlated with the Hodgkin's disease. The 10 per cent incidence of herpes zoster is high, as has been noted by other authors.

SUMMARY

It was possible to separate the lymph nodes from cases of Hodgkin's disease into the histological types of paragranuloma, granuloma and sarcoma. The first type clinically tends to resemble the granuloma in the age incidence and the survival rate, whereas the third type, sarcoma, is clearly a more malignant process, although not necessarily occurring in the older age groups as some have reported. The chances of error in the correct diagnosis of Hodgkin's

TABLE V.

Frequency of involvement of organs by Hodgkin's disease (30 autopsies):

Organ	Percentage involvement	Average weight in grams
Lymph nodes	100%	
Spleen	85	511±480
Bone marrow	75	
Liver	55	1986±509
Lungs	39	
Pancreas	14	
Adrenals	7	
G. I. tract	7	
Kidneys	7	
Gonads	6	
Skin lesions	3	
Thyroid	0	

TABLE VI.

Other diseases present (30 autopsies):

Disease	Number cases	Percentage
Tuberculosis (active)	3	10
Tuberculosis (Healed)	2	7
Herpes zoster	3	10
Influenza-pneumonia	3	10
Treated lues	2	7
Bladder carcinoma	1	3
Diabetes	1	3
Rheumatic heart	1	3
Ulcer (perforated)	1	3

disease are clearly increased when considering the paragranuloma and sarcoma variants.

The detailed structural and cytological variations found in the lymph nodes of cases of Hodgkin's disease were considered and correlated with the clinical findings. Certain trends were detected. Necrosis and the often associated polymorphonuclear leukocytic infiltration were not related to past x-ray therapy (six months or more before) but did occur in patients who subsequently showed a clearly decreased survival time. The occurrence of fibrosis was more of an independent characteristic of the tumor process than a reaction due to x-ray therapy. The degree of fibrosis did not correlate with variations in the survival time. The fibrosis that was present frequently had a peculiar concentric peri-vascular arrangement. Invasion of the capsule which was present in 25 per cent of the cases was not associated with evidence of any greater activity of the disease.

Cytologically, the frequently abundant eosinophils could not be related to clinical characteristics, nor could the Sternberg-Reed giant cells. Langhans' giant cells were rarely seen in the absence of tuberculosis.

Serial passage of filtered lymph node tissue from cases of Hodgkin's disease in embryonated chicken eggs revealed a greater egg mortality. These data are all supportive evidence for the presence of a possible viral agent. The cultures of the lymph node of Hodgkin's disease and control tissues revealed bacteria of various types (but not Brucella) in about 50 per cent of the lymph nodes.

The interchange of Hodgkin's disease with other types of the malignant lymphoma was noted only two times in this series. In both instances the Hodgkin's disease picture was preceded by the pattern of a giant follicular lymphoma. Neither lived longer than the average Hodgkin's disease life expectancy. Two of the Hodgkin's granulomas were sarcoma at the time of the autopsy.

The material from 30 necropsies for Hodgkin's disease was tabulated. The organ distribution was characteristic. The incidence of bone involvement (75%) would probably have been greater with a more systematic survey. Even at autopsy 68 per cent of the spleens and 21 per cent of the livers had weights within normal limits. Tuberculosis was present in 17 per cent of the autopsies, and occasionally produced confusing lesions associated with the Hodgkin's disease process.

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HEREDITARY HEMORRHAGIC TELANGIECTASIA AND RUTIN

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KUSHLIN[1] in 1946 and Cape and Grover[2] in 1948 have each reported a case of hereditary hemorrhagic telangiectasia in which rutin appears to have been beneficial. We wish to add a third case and also to draw attention to abnormal liver function tests found in our patient. A review of the literature reveals a paucity of information concerning the role of the liver in this disease although normal prothrombin times[3, 4] have been mentioned and Gitlow and Frosch[5] have reported a fatal case which at necropsy revealed no hepatic abnormalities.

Hereditary hemorrhagic telangiectasia is a well established clinical entity although somewhat uncommon. Hereditary epistaxis was described by Babington[6] in 1865 and by Rendu[7] in

From the St. Joseph's Hospital Medical Service of Dr. Robert S. Flinn.

1896 who recognized the pathology. Sir William Osler[8, 9] effectively publicised it to the profession early in the Twentieth Century. Since 1931 it has been summarized several times, notably by Goldstein[10] in 1931, Hauser[11] in 1934, Stock[12] in 1944 and Barrock[13] again in 1944. The names of Rendu and Osler are most frequently associated with the condition and hence the term Osler's disease or Rendu-Osler disease.

The accepted diagnostic criteria are: (1) an hereditary tendency, (2) visible typical telangiectasia, and (3) a tendency to bleed from these lesions. A normal clot retraction, platelet count, tourniquet test, bleeding time and clotting time are usually considered essential.

The one etiologic factor of any importance is heredity. At least 80% of cases have antecedents with the disease. There is no sex linkage, the disease being transmitted as a Mendelian dominant equally in both sexes. There is a decreasing severity in succeeding descendency rarely affecting more than five or six generations of the same family.

Pathologically, the lesions consist of dilated capillaries, both venules and arterioles. The walls of the vessels are very thin, often consisting of only a single layer of endothelium covered by a very thin epidermis.[14, 15] The muscular and elastic layers of the vessels are conspicuously deficient.[16] A lesion often disappears after bleeding due to thrombosis. The most common sites of lesions are the naso-oral cavities usually Kiesselbach's triangle and the inferior surface of the tongue. Frequent additional sites are the ears, face, scalp, conjunctivae, fingers and occasionally toes. The lesions may occur anywhere in the gastro-intestinal tract[17] and have been described in the brain, liver, spleen, kidneys and uterus. The latter locations have a more serious prognostic significance and patients with the disease should always be considered as potential internal bleeders.

Treatment has been unsatisfactory. It has usually been directed toward destruction of the lesions, transfusions and general symptomatic care. The usual regimen consists of:

1—Mechanical nasal packs.
2—Snake venom (Agkistrodon, Crotalus, Vipera and Notechis)
3—Local: a Radium and X-ray.
b Thermal - galvanocautery and electrocoagulation.
c Chemical - chromic acid beads, carbon dioxide snow and urethane solutions.
d Surgical - sub-mucous resection.
4—Blood transfusions.
5—Symptomatic - iron, calcium gluconate, vitamin K and vitamin C.

Rutin, in view of the reported successes[1, 2] should perhaps be added to the list. Certainly it is deserving of further trial. Chemically, it is a glucoside derived from certain plants, notably buckwheat leaf. It is closely related to ascorbic acid, is non-toxic in man and has been found in preliminary trials to increase capillary resistance in a number of hemorrhagic conditions. One report[19] has indicated its efficacy in reducing the incidence of hemorrhage in hypertension, the capillary fragility in drug reactions and the bleeding tendency in other poorly defined hemorrhagic states.[20]

The prognosis as to longevity in hereditary hemorrhagic telangiectasia is generally good although an occasional victim succumbs to hemorrhage. Mortality is estimated at four per cent and of these more than half approach the biblical "three score and ten."

REPORT OF CASE

H. D., a 65 year old white male of mid-European extraction entered St. Joseph's Hospital November 8, 1948 with the presenting complaint of "nosebleeds." From infancy until age 35 he had almost daily nosebleeds, none severe and usually stopping spontaneously after a few minutes. They occurred spontaneously or with trauma.

After the age of 35 the nosebleeds became more severe. In 1936, at the age of 54, a series of cauterizations of the nose were done which gave no relief. In 1938, several injections of "moccasin snake venom" were tried without relief. Shortly thereafter, vitamin K was also tried with similar results. From 1943 to 1945 the patient had four widely spaced episodes of bright red bloody vomiting. The emesis was on each occasion followed by passage of dark red blood in stools. Peptic ulcer was suspected but never proven. Since 1946, the epistaxis continued almost daily until April 1948. In April, he was started on rutin, 60 mg. daily and ascorbic acid, 100 mg. daily. Both were soon increased to 100 mg. three times a day. Since that time there has been a generalized improvement characterized by increased strength and energy, marked decrease in number and severity of the nosebleeds and a greatly reduced number of "red spots on hands and face." He still has spontaneous epistaxis with considerable loss of blood occasionally but is generally enthusiastic about rutin. For about one and one-half years

before beginning rutin therapy the patient had been unable to work because of general debiilty. However, since August 1948 he has been working regularly without distress.

Past medical history was not significant.

Family history revealed that his father died at 62 from an indefinite cause but was a "bleeder." His mother died at 60 from heart disease. One brother died at 69 from "bleeding." One paternal uncle was a "bleeder." Two other brothers are living and well at 70 and 72. Two sisters are living and well and each has one son who is a "bleeder." The patient was careful to specify that by "bleeder" he meant frequent nose bleeds.

Pertinent physical findings revealed a well developed and nourished, somewhat pale male of stated age 65 in no distress. His skin was generally pale, dry and warm; it was stigmatized by several small, round, purple, spider-like, raised spots on both ears, cheeks, finger tips and upper thorax anteriorly which blanched with pressure. There were similar telangiectatic lesions in Kiesselbach's triangle of the nose and also on the inferior margin of the tip of the tongue. The abdomen was not remarkable except for the liver, which extended 3 cm. beneath the right costal margin and was smooth, slightly tender and with a rounded edge.

Laboratory:

1—Urine: normal
2—CBC: 8.5 Gm. Hb., 4,100,000 RBCs, 6200 WBCs with a normal differential distribution.
3—Coagulation time 3 minutes.
4—Bleeding time 1 minute.
5—Platelets, 150,000/Cu mm.
6—Prothrombin time 100%.
7—Bromsulphthalien test showed 45% retention after 30 minutes.
8—Cephalin-cholesterol floeculation was two plus at 24 hours and three plus at 48 hours.
9—Hippuric acid test was 87 milligrams elimination.
10—Rumpel-Leed test showed an absence of petechiae after 15 minutes.

COMMENT

A brief review of the literature concerning hereditary hemorrhagic telangiectasia is reported together with the use of rutin as a possible therapeutic agent. A case is summarized describing the course of the disease before and after the use of rutin. Mention is made of the apparent hepatic dysfunction existing in this patient. It is hoped that interest will be stimulated for additional trails of rutin and attempts made to correlate liver function with the disease.

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ACUTE INFECTIOUS LYMPHOCYTOSIS
Report of Four Cases in a Family

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PAUL and Bunnell[1] in 1932 described the heterophile agglutination test which, when positive, is practically diagnostic of Infectious Mononucleosis. Occasionally patients are seen who fail to develop high titers of heterophile agglutinins in the blood, although the blood studies otherwise and the benign course of the illness suggest Infectious Mononucleosis. Many of these cases fall into the disease pattern of Infectious Lymphocytosis, as described first by Smith in 1941. In his publication the distinguishing features were pointed out which separated this disease from Infectious Mononucleosis[2]. This differentiation is still not too widely appreciated or recognized.

The blood findings of acute Infectious Lymph-

ocytosis, when accompanied by extreme elevation of the lymphocytes and a negative Paul-Bunnell reaction, may be temporarily confused with the blood picture of chronic lymphatic leukemia. Meyer[3] has clearly summarized the comparative features of these diseases.

The symptom complex of the disease has usually been described as being mild and consisting of pallor, fever, irritability, and at times paraumbilical pain and mild joint discomfort. Skin rashes, jaundice, splenomalgy, pharyngitis, and lymphadenopathy are not usually observed in Infectious Lymphocytosis. Thelander's and Shaw's[4] report indicates that the central nervous system may at times be involved. Two of their patients had elevated lymphocyte counts in the spinal fluid (40 and 70 cells). These patients had fever, convulsions, and muscular twitchings. No fatal cases of Infectious Lymphocytosis have been reported. The treatment to date is purely symptomatic.

The total leukocyte count in the peripheral blood varies markedly; from 20,000 to 120,000 per c.mm. The lymphocytes are small or moderate in size; and, unlike the cells in Infectious Mononucleosis, there are few or no fenestrations to be seen in the nucleus. The cytoplasm takes basophilic stain and is without the "foamy" appearance of the "typical cells" of Infectious Mononucleosis. The number of eosinophils, unlike Infectious Mononucleosis, may be increased in number early and during the course of the disease. The blood findings usually return to normal within two to eight weeks. Bone marrow studies, in the typical cases reported by Smith, have revealed 30 to 43 per cent of the nucleated cells to be lymphocytes[2, 5].

The infectious nature of the disease is emphasized by the reporting of four cases that occurred simultaneously in four of five siblings. This family lived on a comparatively isolated farm.

CASE No. I.

A female, aged 11 years, was seen by one of us (R. S.) on the 22nd of May 1947. She complained of generalized abdominal pain which had been present, intermittently, for two or three weeks. The pain was vaguely localized in the lower abdomen at the onset of her illness, but later became more intense under the left anterior costal margin. At the onset of her illness she experienced some mild nausea, but no vomiting. During the preceding ten days she had expelled five to six formed stools each day. The stools were normal in color, without blood. There were no other complaints except for a mild generalized headache and a cough of one week's duration. There had been no skin rash or sore throat. She remained active during her illness and was never confined to bed. Past history of childhood diseases consisted of chicken pox and measles. She had not been exposed to or had pertussis.

Examination revealed a rather pale, well-developed and well-nourished child whose temperature was 99°F. The skin was clear, and there was no local or generalized lymphadenthere was n olocal or generalized lymphadenopathy. Eye examination revealed normal extraocular motions; the pupils were round and equal, and reacted actively to light and accommodation. The ocular fundi were normal. The mouth, gums, and pharynx appeared to be normal. The heart and lungs were normal. The abdomen was normal in contour; the liver and spleen could not be palpated. There were no localized areas of tenderness or muscular spasm. The extremities were normal, and the reflexes were equal and active.

The blood Kline reaction was normal. The urine was normal. Examination of the stool was negative for blood; giardia lambia ova were present. The blood studies are outlined in Fig. I.

The other children in the family and both parents were then examined.

CASE No. II.

M. J., Male, aged 4 years, was examined on the 14th of May, 1947. His mother said he had complained of abdominal pain that had been present off and on for three or four weeks. The pain was chiefly in the mid-abdomen. Nausea and vomiting was absent. For a week he had passed an occasional liquid stool, but no blood. He had not had fever, sore throat, or skin rash.

Fig. I.

Date	Hbg. Gms.	RBC cmm.	WBC cmm.	Paul Bunnell	Differential Count in %						
					Lymphocytes			Neut.	Eos.	Mono.	Baso.
					Small	Med.	Total				
5/14/47	12.2	4,120,000	40,800		39	31	70	19	11		
5/16/47				1:28	67	2	69	26	5		
5/22/47	10.15	3,850,000	13,380	1:28	66		66	28	5	1	
6/ 4/47			11,450	1:112	50	1	51	46	2	1	
1/10/48	10.45	3,750,000	9,450		38		38	56	2	3	1

It will be noted that the eosinophil count returned to normal as the disease subsided.

Fig. II.

Date	Hbg.	RBC cmm.	WBC cmm.	Paul	Differential Count in %						
	Gms.			Bunnell	Lymphocytes			Neut.	Eos.	Mono.	Baso.
					Small	Med.	Total				
5/14/47	10.45	3,690,000	26,350		6	68	74	16	9		1
5/16/47			26,950	1:28	62	11	73	23	3	1	
5/22/47	10.8	3,750,000	9,000		53		53	37	9	1	
6/ 4/47			9,150		36	2	38	49	7	6	
1/10/48	11.2	4,420,000	8,000		35		35	56	1	8	

Past infections included measles and chicken pox, but not pertussis.

Physical examination revealed a pale child with clear skin. The temperature was 98.4°F. There were a few small non-tender, firm, posterior cervical and axillary lymph nodes. The teeth, gums, and pharangeal wall appeared to be normal. The heart and lungs were normal. The abdomen was normal in contour. The spleen and liver were not palpable; and local tenderness and spasm were absent. The extremities, genitalia, and rectum were normal.

Laboratory examination: The blood Kline reaction was normal. Urinalysis: The urine was normal. The stool contained giardia lambia ova, but was negative for blood. The blood findings are outlined in Figure II.

CASE No. III.

A. J., female, aged 9 years, was examined on May 22, 1947. She had complained of vague abdominal pain and some pain in the right knee for one week. The abdominal pain was chiefly limited to the right upper quadrant, along the costal margin. Nausea, vomiting, and diarrhea were not present.

Over the same period of time she had experienced a mild cold without fever. The knee had never become swollen or tender to touch, but she had complained of aching in the joint after the day's activities.

Her past history was irrevelent. She had had chicken pox and measles, but she had not had pertussis.

Physical examination revealed a fairly well-developed and well-nourished child without acute distress. The skin was clear and of normal color. The lymph nodes were not enlarged. The temperature was 99°F. The pharyngeal wall appeared to be normal. The eyes and nose were normal. The heart and lungs were normal. The abdomen was normal in contour and not tender. The liver and spleen were not palpable. The genitalia and rectum were normal. The extremities were normal. There was no limitation of motion. The knee joint was normal to inspection. The blood findings are outlined in Figure III.

CASE No. IV.

P. J., female, aged 2 years, was examined on May 16, 1947. Her mother stated that for the past one to two weeks she had occasionally complained of abdominal pain, without nausea, vomiting or diarrhea. She had not complained of a sore throat, and appeared otherwise to be in good health. She had had none of the usual childhood diseases.

Physical examination revealed a normally well-developed and well-nourished child without abnormalities. The temperature was 98.6°F. Complete physical examination was normal. The liver and spleen could not be felt, and there was no enlargment of the peripheral lymph nodes.

Laboratory examination: The blood Kline reaction was normal. The urine and stool were normal. Occasional WBCs were in the urinary sediment. Stool specimen was negative for blood and parasites. The blood examination revealed the findings outlined in Figure IV.

DISCUSSION

The blood smears of these patients were examined by Dr. William Dameshek, who felt that in view of the doubtfully positive heterphile agglutinations the disease represented was probably Infectious Lymphocytosis[6].

The mother, aged 37 years; the father, aged 40 years, and a child, aged 4 years, had blood counts made while the other members of the family were ill, and their blood studies were normal on two separate occasions.

Finucane and Philips[7] in reporting twenty-one cases point out that the two sexes are about

Fig. III.

Date	Hbg.	RBC cmm.	WBC cmm.	Paul	Differential Count in %						
	Gms.			Bunnell	Lymphocytes			Neut.	Eos.	Mono.	Baso.
					Small	Med.	Total				
5/16/47			37,000		53	12	65	24	10		1
5/22/47	9.15	3,510,000	25,100	1:7	77		77	16	6	1	
6/ 4/47			18,350	1:56	57	6	63	35	1	1	1
1/10/48	11.2	4,000,000	6,450		37		37	55	2	2	2

equally affected with this disease. They believed that a Paul-Bunnell reaction with a titer of 1.56 to 1:112 are questionably positive, while reactions of 1:160 and over are definitely positive for Infectious Mononucleosis. One of our patients, Case I, had a doubtful reaction (1:112). It would appear unlikely that both diseases are represented in this series, and we feel they are best explained by Infectious Lymphocytosis. The following features help to differentiate the disease presented: the extreme elevation of the white cell count; the increase in the eosinophil count; and the absence of fenestrated nucleus and "foamy" cytoplasm seen in Infectious Mononucleosis. Clinically the absense of splenomegaly, pharyngitis, and lymphadenopathy help to establish the diagnosis.

Elevations of the lymphocyte count to the degree often seen in this disease may suggest chronic lymphatic leukemia, especially in the presence of a negative heterophil antibody test. Therefore, other children in the family, even those that appear normal, should have blood studies made to determine the presence of Infectious Lymphocytosis. This precaution will at times aid in the proper diagnosis and prevent unnecessary anxiety.

The low hemoglobin and red blood cell count present in each of these cases, we feel, were not greatly altered from the normal variations of children.[8] These patients were not treated, and the hemoglobin determinations remained about the same in all cases over the period of this study.

SUMMARY

1. Four of five children in one family presented the blood picture of Infectious Lymphocytosis.
2. The contagious nature of this disease may at times aid in the differentiation of this disease from lymphatic leukemia.

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Fig. IV.

Date	Hbg.	RBC cmm.	WBC cmm.	Paul	Differential Count in %						
	Gms.			Bunnell	Lymphocytes			Neut.	Eos.	Mono.	Baso.
					Small	Med.	Total				
5/16/47			64,400		80	3	83	13	3		1
5 22/47	10.8	3,770,000	50,850	1:14	82	2	84	13	2	1	
6/ 4/47			49,500	1:28	91	1	93	4	3		
1/10/48	9.4	3,450,000	7,200		31		31	65	2	1	1

Arizona Medical Problems

CONSULTATION AND CASE ANALYSIS

ARIZONA MEDICINE again presents an unsolved and difficult case from the practice of Arizona physicians, with the Case-Analysis and comments of a specially-chosen and nationally-known Consultant.

Any physician who has an undiagnosed case which has defied other methods of solution may send it for consideration. The case should be completely worked up, but an editor will help compose the report. Whenever the need for an answer is urgent, the Consultant's reply will be sent direct to the submitting physician, before publication.

Please send communications and data to Dr. W. H. Oatway, Jr., 123 S. Stone Avenue, Tucson, Arizona, or care of The Editor, Arizona Medicine.

The current case is one of interest to general practitioners, internists, chest specialists, and thoracic surgeons. It presents *the modern diagnosis and management of spontaneous pneumothorax.*

This case was sent to Arizona by a surgeon in the mid-west, and it was returned to him later for care. By chance this surgeon happens to be a prime authority on management of such cases, and the use of *poudrage therapy.* By intent we have asked him to discuss this case, his handling of the problem, and the condition in general.

The CONSULTANT is *Dr. John D. Steele* of Milwaukee, Wisconsin.

Dr. Steele is Thoracic Surgeon for the Milwaukee County Sanatorium; Assistant Clinical

Professor of Surgery at Marquette University; and Consultant in Thoracic Surgery for the Veteran's Administration Hospital at Wood, as well as for several regional county sanatoria.

He is a member of the American Association for Thoracic Surgery and the American College of Surgeons. He is currently president of the Mississippi Valley Trudeau Society, a former vice-president of the American Trudeau Society, and chairman of the potent committee on Therapy for the latter group. He is a diplomate of the Board of Thoracic Surgery.

Dr. Steele is known to many Arizonans, having visited here often and having addressed the charter meeting of the Arizona Trudeau Society in 1946.

* * *

CASE NUMBER XVII

The patient is **a white male aged 70 years.** He was sent to southern Arizona to avoid the rigors of a Wisconsin winter, and arrived during December.

The **chief complaints** were dyspnea, symptoms of chronic bronchitis, the fear of recurring spontaneous pneumothoraces, and an acute grippe-like "cold" which had begun before he left the mid-west.

The **history of his present illness** began about **five years ago,** when he abruptly became very short of breath, had pain in the left lower chest, and **was found to have a spontaneous pneumothorax at the left apex and base.** Symptoms of a chronic bronchitis began at that time and have persisted since.

The **past medical history** is mentioned at this point. He had pulmonary tuberculosis when a young man, but it had long since healed and ceased to give him any trouble. He had a gastric resection for ulcer in 1917, followed within a year by a gastro-enterostomy. His **occupation** has been that of an official in a manufacturing concern, and he has led a sedentary life. No pertinent family history was recorded, and the patient did not use tobacco or alcohol. He did not know of any sputum or blood tests.

Progress following the first spontaneous collapse included a slow re-expansion of the left lung, and occasional episodes of pleurisy in the next few years. **A year ago,** in May **the patient had a sudden attack of dyspnea and pain in the left chest,** and Dr. Steele was called. The patient was found to be shocked and very cyanotic, and there were signs of a pneumothorax at the left base. **A needle was inserted,** and air (under positive pressure) was allowed to escape. He was taken to a hospital, where he had a stormy convalescence, due to the dyspnea which was aggravated by even a small residual pneumothorax. **Repeated decompressions** were required over a period of several weeks, using a needle inserted into the chest through a rubber plaque, and attached by tubing to a water-seal bottle. The small **fistula finally closed** and the lung expanded completely.

Three months later another left basal pneumothorax occurred, and the lung re-expanded after only one decompression. Two similar episodes occurred, however, during the following month, with response to deflation as before.

The condition then remained stable for the next two months and **it was decided to have the patient go to Arizona** to avoid the effects of acute respiratory infections, which had already begun with the winter weather. His case-report and suggestions were sent ahead, and care was arranged for him in several cities along the route in the event that he had trouble on the train.

On his arrival in Tucson it was found that he had a subsiding acute bronchitis, with mild cough, expectoration of 2 teaspoonsful of mucopurulent sputum each morning, and moderate dyspnea and wheezing. He had no fever, there had been no pain in the chest for weeks and he had no sinus infection. He had a mild anorexia, was 20 pounds underweight, but there was no other systemic complaint. He was thin (115 pounds); the BP was 145/85; the pulse was 100/min; the nail-beds were quite cyanotic; the chest was emphysematous in contour and mobility; and there were no rales or friction-rubs. A blood count found 3,940,000 RBC., and 11 gm. (80%) of Hb.

X-rays of the chest, taken during the previous 6 months, showed a notable pulmonary emphysema; numerous old fibrous scars and calcifications in the upper half of each lung field; and a dilated ascending aorta with aortic sclerosis. There was no pneumothorax present. **Fluoroscopy** showed no new finding except a tenting of the right diaphragm, and confirmed the absence of any intra-pleural air or fluid.

It was decided to observe the patient, to continue use of KI he had been taking, and to keep oxygen and a sedative-expectorant mixture at hand for PRN usage.

Two weeks later the patient developed a fever of 100 degrees, an increased dyspnea and cyanosis, pain in the **right** lateral chest area, purulent sputum which contained pink and rusty clots, and a few fine rales at both posterior lung bases. He was sent to the hospital, and oxygen and sulfamerazine were prescribed. An x-ray of the chest showed an odd, **fibrous-appearing peribronchial pneumonia extending from the right hilum to the base.** During the next few days, **a pocket of pneumothorax appeared** above the dome of the diaphragm medially, and a fluid level was seen when he was fluoroscoped.

Evidences of infection cleared rapidly, but he required oxygen much of the time for 10 days and a pocket of air (similar to a pleural bleb) persisted at the time of his discharge to a rest-

home. There was a to-and-fro friction-rub at the anterior right base, and medium-coarse rales and rhonchi were heard at the right base and hilum. Sulfamerazine, an ephedrine compound, a liver and iron compound, and oxygen (PRN) were continued. The pneumothorax area became solid to percussion (fluid), he gained 12 pounds, and his blood count improved.

During the next six weeks the patient had **two episodes of right basal pain,** but improved in general and was able to take short walks. Following one walk he had pain at the **left** lung base, but there were no other symptoms; fluoroscopy showed the lungs to be similar to the last hospital x-ray, with a residual truncal accentuation, a pleural fibrosis, and the "bleb" at the right base.

He returned to Milwaukee after four months in Arizona. It was later heard that a month or so after his return he had had **several episodes of left basal spontaneous pneumothorax**, and that **an attempt was to be made to produce pleural symphysis.**

QUESTIONS

1. What therapy was used later in this case? Was it effective?
2. What are the usual causes of spontaneous pneumothorax at various ages? What was the cause in this case?
3. What are the methods of producing obliteration of the pleural space? Which is most effective?

M. D., Tucson

CASE-ANALYSIS AND ANSWERS BY DR. STEELE

1. *The therapy* which was used in the case of this elderly man was an obliteration of his left pleural space by the use of poudrage, as will be described. There had been a frequent recurrence of the spontaneous pneumothoraces, and any amount of collapse had produced an uncomfortable and dangerous limitation of his respiratory function, due to the emphysema and fibrosis.

The procedure was effective, in that he had no further recurrences. He died three years later in California, at the age of 74 years, but the exact cause is unknown.

2. *There are several causes of spontaneous pneumothorax.* In the younger age groups, the most common cause of a spontaneous pneumothorax is a congenital weakness of the periphery of the lung. A series of alveoli rupture and the air collects beneath the visceral pleura forming a bleb. When this bleb ruptures, the air escapes into the pleural cavity. The next most common cause is pulmonary tuberculosis. Another less frequent cause: localized obstructive emphysema due to a foreign body such as a piece of peanut. In the *older groups, the most frequent cause is the rupture of an emphysematous bulla. This was the cause in the present case.*

3. *Many agents have been used experimentally and clinically in attempts to obliterate the pleural cavity by the production of adhesions.* My own experience has been limited to the following: blood, saturated glucosa solutions, talc suspensions in distilled water, insufflation of plain and iodized talc. Blood, glucose and talc suspensions were found to be unsatisfactory, as our results with them were far from uniform. Talc insufflations were found to give uniformly good results if sufficient powder was used (8 to 10 gm.). The addition of iodine to the talc has been considered unnecessary.

Three methods of powder insufflation are available:

(a) Insufflation through a *large bore needle* with a powder atomizer.
This is useful when the pneumothorax space is small.

(b) Introduction of a thoracoscopic *trocar and cannula* into the pneumothorax space under local anesthesia and insufflation of powder through a catheter. This has the advantage of allowing inspection of the surface of the lung prior to insufflation, and inspection following insufflation to insure even distribution of the powder.

(c) *Thoracotomy* under general anesthesia with the removal of a small segment of rib. The advantages of this method are: first, freedom from pain which is often severe as soon as the insufflation is started; second, the surgeon has the opportunity to rub the parietal pleura with dry gauze which, in itself is a satisfactory method of producing pleural adhesions; and, third, inflation of the lung by the anesthetist at the conclusion of the procedure so as to bring the pleural surfaces into apposition. We now prefer this method when not contraindicated.

Before using any method of powder insufflation, *it is essential that the lung be fully expanded,* indicating closure of the broncho-pleural fistula. The reason for this is that it is essential after powder insufflation that the pleural surfaces be brought into immediate apposition —otherwise obliteration of the pleural cavity

will not be effected. In methods (a) and (b) it is necessary that a preliminary artificial pneumothorax be induced prior to insufflation. In method (c) this is not necessary.

4. *In the case which has been described, a needle* was inserted into the pleural cavity and a small pneumothorax induced. Even this small pneumothorax produced considerable dyspnoea as the patient's respiratory reserve was extremely low. A larger needle was immediately introduced and about 3 grams of talc insufflated into the pleural cavity. All air was immediately withdrawn. As is the rule in this type of procedure, the patient experienced considerable pain for 24 hours but had no other reaction.

It should be emphasized that *obliteration of the pleural cavity is not advocated in every spontaneous pneumothorax,* but only in those which cause considerable disability due to recurrences. It should also be re-emphasized that poudrage is not a treatment for non-expanded lungs following spontaneous pneumothorax but is used only to prevent recurrences. The treatment of non-expanded lungs is another subject entirely and often consists of the excision of the broncho-pleural fistula or lobectomy.

John D. Steele, M. D.,
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(Note:—Dr. Steele's report on poudrage and obliteration methods appeared as follows: "Production of Pleural Adhesions for Therapeutic Purposes," American Review of Tuberculosis, 55:299, October, 1947.)

ARIZONA MEDICINE
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Editorials

Poliomyelitis

Comes summer and fall, and one needs to recall the *picture of poliomyelitis.* In general, children are still most commonly affected, but more cases are seen in teen-agers and adults than formerly. Many people have the subclinical type of infection; in some the recognizable disease occurs; but very few develop paralysis, or die. No more definite than in 1948 is the method of spread, a protective vaccine, or a specific therapy.

Three stages of the clinical picture may be noted,—(1) *the systemic phase* begins after an incubation period averaging 3 to 10 days. The symptoms are those of an acute, moderate, general infection, with headache (frontal), nausea, an unstable colon, fever (100-101 degrees), mild pharyngitis; they last 24 to 36 hours. Any diagnosis in this stage is presumptive, and can only be based on a history of contact. Most patients (about 80 per cent) have no further symptoms.

(2) About 20 per cent of those infected progress, after a lag period of 2 to 4 days, to the *central nervous system phase.* The same symptoms recur, though slightly accentuated, and in addition one sees drowsiness, irritability, emesis, anxiety, sweating on the face and neck, and hyperesthesia of the skin as evidence of the *early* CNS stage. Progression to a *late* CNS phase is indicated by "stiff neck," the "spine sign," "head drop," and tremors in the extremities.

(3) The third phase is *paralytic,* with some degree of weakness and some amount of flaccid paralysis of one or more muscles or muscle groups. Pain and spasm are inconstant.

In many patients the systemic phase is not noted, and the illness begins with the CNS phase. In a few patients the first evidence of illness is the paralytic phase.

The *clinical types* of poliomyelitis are the nonparalytic (including the abortive), and the paralytic, which is subdivided into spinal, bulbar,

spinobulbar, and the less common encephalitic, meningitic, and cerebellar types.

Lumbar puncture is of value in the late CNS stage; an earlier puncture may obscure subsequent spinal fluid findings. Pressure is normal or increased; the fluid may be clear or hazy (depending on the cell count); cells usually range between 15 and 500; the early cells are PMN leucocytes, the late cells are lymphocytes; protein is somewhat elevated (above the 15 to 45 Mgm. normal); sugar is normal (50 to 80 Mgm.); bacterial studies are negative.

Unless a general practitioner has special training in diagnosis and care of the disease, everyone concerned will usually profit by a consultation with a skilled internist or orthopedist—and quickly. The *Arizona* polio season usually reaches its peak in late summer and early fall—the latest season, along with California, of any area in the United States.

To mix a metaphor, let us hope that our eyes are alert and our wicks are trimmed.

A. M. A. Educational Campaign

During the planning and formative stages of the A.M.A. Educational Campaign, many indications have been given that it is well planned and will be well executed.

A few doctors have expressed disappointment that an immediate flood of publicity did not issue forth from Chicago, and that wide-scale advertisements did not appear in newspapers and on the radio. However, the fact that the campaign seems to be progressing slowly is evidence that every detail of the program has been meticulously studied, and that every organization which will participate in it has been assigned an appropriate part in the coordinated whole.

It would have been easy to produce a sudden flash of hastily manufactured propaganda. This would have had a sensational impact, but would not have had any lasting effect.

The A.M.A. has very wisely adopted a soundly conceived plan which will move comparatively slowly, but because of this will be the more powerful. Its effect will be non-sensational, and therefore long lasting.

The literature and other items of campaign material which the A.M.A. is producing seems slow in making its appearance. One reason for this is that all the campaign writing is being checked and re-worked to produce the most compact and highest quality material possible. A second reason is that all of it is being produced in lots of tremendous size. Most of the printing orders add up to many millions of copies.

Early in the campaign the fundamental plan was announced by the A.M.A. to the component state associations. This has allowed the state and county societies to develop their own programs in such a way as to dovetail into and complement the national campaign.

The A.M.A. campaign has been set up and planned with two major characteristics.

First, it has been stressed from the beginning that it is an affirmative campaign. Doctors know that compulsory health insurance will produce poor medicine. The public should be told this and should be told why. However, the best way to prevent the adoption of compulsory insurance is to offer something better. Sound voluntary health insurance is the answer.

The 12-point program of the A.M.A. is another part of the affirmative approach which has been used in the campaign.

Most of the A.M.A. 12-point proposals, and voluntary insurance, are part of a gradually developed A.M.A. plan for the improvement of medical care. The fact that federal health insurance has become a political issue has highlighted the A.M.A. program, and the educational campaign will highlight it even more.

The assignment of high priority to the affirmative aspects of the plan is especially fortunate. Achievement of these objectives will not only defeat the drive for compulsory insurance this year, but also will make less likely the instigation and chances of success of future moves toward socialized medicine.

Secondly, the A.M.A. campaign has been designed as a broad, public campaign.

One advantage of the widespread publicity which has resulted from A.M.A. announcements has been that a large number of lay people who are interested in free practice of medicine have been aroused and have become aware of the seriousness of the situation.

The A.M.A. campaign will continue to inform the people concerning the dangers of state medicine. At the same time they will be informed concerning the advantages of the plan which the medical profession is sponsoring.

Public opinion polls which have been reported in the daily press indicate that a large propor-

tion of the people, when asked to state their views on socialized medicine and government health insurance, answer by saying that they do not know enough about the question to have an opinion. The need for stating facts through a widespread educational campaign is very evident.

If the people are acquainted with the type of medical care which is offered by government medicine; if they are cognizant of its cost, and realize its effect on medical progress; and if they understand the rationale of the A.M.A. program for the improvement of medical services, there can be no doubt as to the outcome.

TOPICS OF CURRENT MEDICAL INTEREST

REPORT OF THE DELEGATE

House of Delegates, American Medical Association, June 6-10, 1949

(Continued from July Issue of Journal)

In addition to the usual routine employed by the House of Delegates in the dispatch of its business, that of receiving reports from its Officers, Board of Directors, Councils and Committees, the election of Officers, making necessary changes in the Constitution and By-Laws of the A. M. A., or the Code of Ethics when deemed advisable, it receives and considers many resolutions. Often these refer to the ramifications and problems in present day socio-economics of medical practice.

For instance, at this Session, the House amplified the A.M.A. twelve point program to include specific details relative to financial aid to medical schools, in which it said in part: "The A.M.A. would prefer to see medical schools receive the support they require from private philanthropy or local public funds. Unless and until such support is provided, it may be necessary for some medical schools to accept financial aid from the Federal Government. Such aid, however, must carry with it the assurance of freedom from political control . . . .".

In connection with the expansion of the twelve point program also, the House made possible further emphasis upon the extension of voluntary health insurance programs, and set the machinery in motion for a doctor-layman conference to be held sometime this fall, calling in representatives from farm, labor and business groups for a full discussion of the ways and means to attain an ultimate objective in the enrolment of many more millions of our people in voluntary methods of protection against hospital and medical obligations.

Another significant move was the adoption of a resolution which commands the Board of Trustees to "Create a special committee, to be composed of representatives of the Board of Trustees, of the House of Delegates, and other Fellows of the A.M.A., whose function it shall be to arrange a conference with members of Congress, with a view of developing legislation that would meet the objectives of the American Medical Association program in its efforts to solve the problem of making medical care more readily accessible to the American People." It is hoped that this Committee can help us cooperate with Government in fields of health care or prevention in which we all agree. An action committee of this sort should be able to call periodic conferences with Congressmen to develop health legislation which will incorporate some of the thinking developed by American Medicine.

Perhaps the most important piece of constructive legislation passed by the House was the adoption of a set of principles for medical societies to use in approving lay-sponsored medical care plans. The principles had been endorsed prior to the annual meeting with committees representing the Council on Medical Service, and officials of the Cooperative Health Federation of America, including the co-ops, union, farm and community groups. It should be emphasized that these twenty principles are not mandatory upon any state or county medical society, but should be used as a guide to these local organizations in determining the eligibility of any such plans for approval.

By approving these principles also, the House of Delegates recognized that local areas may find it necessary to make modifications in dealing with the medical or professional care of these groups, and that any such plan requesting A.M.A. approval must first be endorsed by a

State or County Medical Society. This set of principles should and will support the various medical societies in judging the acceptability of all lay-sponsored programs, as well as strengthening their case when it becomes necessary to oppose unacceptable legislation as these groups endeavor to engage in the corporate practice of medicine through legislative channels. Above all, the principles will aid the medical societies in their efforts to protect the public who participate in these lay-sponsored plans.

Among the twenty principles, the following five are predominant:—

1. Any licensed physician (M.D.) in the community who meets the plan's professional standards, and who wishes to participate shall be allowed to render professional care to the participants. Within reasonable limits, plan members shall be given free choice of participating physician.

2. The membership of the governing body of the plan shall include representatives of the medical profession.

3. The plan shall be non-profit, paying no dividends to beneficiaries, or others.

4. The plan must provide each member with a written statement of the exact services to be provided, the conditions, exclusions, and dues to be paid.

5. The plan shall comply with the A.M.A. Principles of Medical Ethics,—particularly with that section barring exploitation of the doctor's services, "for the financial profit of the agency concerned."

Other items of interest to the membership, concerning which definite action was taken included:—

1. Directed the Council on National Emergency Medical Service to draw up plans for the selection of ASTP and V-12 trained physicians, when and if it becomes clear to the Council that the essential requirements of the military medical services cannot be met through voluntary enlistments.

2. Expressed disapproval of the extension of so-called "Social Security" to self employed individuals, including physicians, as embodied in a piece of legislation now before Congress.

3. Passed a resolution requesting the A.M.A. to seek amendment to the federal law on workmen's compensation, to the effect that all covered federal employees be granted free choice of physicians for injuries sustained in line of duty.

4. A resolution was introduced concerning the limitation of the Government of the United States' activities in engaging in any business, professional, commercial or industrial enterprises, in competition with its citizens, except as specified in the Constitution. The House of Delegates approved the resolution in spirit, realizing, of course, the difficulties of its accomplishment.

5. The House gave endorsement to a proposed Code of Ethics to govern the World Medical Association. This endorsement was in order since the A.M.A. is a member organization.

6. Upon recommendation of the Council on Scientific Assembly, the House gave approval to the creation of a new Section, that of Section on Physical Medicine and Rehabilitation.

7. A seat for a new Delegate was made. An invitation was extended to the newly created Department of Medicine and Surgery of the U. S. Air Corps to name one Delegate, to sit in the House with representatives of four other government agencies, namely, the Army Medical Corps, the Navy, Veterans, and United States Public Health Service.

8. Other matters disposed of at this Session included: the approval of additional changes in the A.M.A. Code of Ethics, in an attempt to remove many of the ambiguities in the old Code; directed the Board of Trustees to study and find some practical methods of curtailing V. A. hospital care for thousands of non-service connected cases now being admitted at tax payers' expense, and endorsed recommendations dealing with the placement of graduates of foreign medical schools; ordered the Council on Medical Education and Hospitals to make a survey of all foreign school qualifications for recommendation to all State Examining Boards. The House, furthermore, accepts certain recommendations of the Council which is attempting to solve the "interne and resident" hospital distribution problem, as well as promoting better training, with emphasis upon general practice for recent graduates of medical schools.

The Interim Session of the House of Delegates will be held in Washington, D. C. December 6 to 9, this year, while the Annual Session moves to San Francisco in 1950.

Respectfully submitted,

Jesse D. Hamer, M. D.
Phoenix, Arizona.

RX, DX, AND DRS.

By Guillermo Osler, M. D.

In June of this year we tut-tutted the writers who were rushing the discoverer of NEOMYCIN into an early announcement. . . . Now, in July, Dr. Waksman is defending himself nicely—and so is neomycin. . . . William Laurence, the accurate science editor of The New York Times, describes the report which Dr. Gladys Hobby (of the biological laboratory of Charles Pfizer & Co.) gave to the New York Academy of Sciences on June 25th. . . . Enough neomycin has now been manufactured to be used in animals. It has been found to successfully suppress tuberculosis in mice. It is non-toxic in large doses. It produces resistance only one-fiftieth as often as streptomycin; the resistance develops much more slowly; the resistant strains are apparently not the same ones; and neomycin is effective against streptomycin-resistant strains! . . . Applying the dosage data to humans, it is expected that they will be able to tolerate amounts far in excess of those required to control the disease. . . . Neomycin is probably made up of several fractions, and Dr. Waksman and colleagues are attempting to find the most potent one. . . . Maybe, perhaps, possibly we might legitimately uncross one pair of fingers.

St. Mary's Hospital, the Tucson Medical Center, and the Pima County General Hospital have been the RECIPIENTS OF A MOST UNUSUAL GIFT. Mr. Gerry Pierce of Tucson, a famous etcher, water-colorist, and teacher, has made "lifetime loans" to those institutions of more than fifty of his water-colors, and St. Luke's Sanatorium will probably also benefit. . . . The pictures are to be placed in rooms ordinarily used by patients with chronic illnesses, and the locations are to be changed at intervals. Mr. Pierce has lectured at St. Mary's on the effect of line and color on patients, and the St. Mary's staff intends to make observations on this subject in future years. . . . It is a thoughtful and generous gesture.

AMINO ACIDS are a prime source of nitrogen. They are more effective when given orally than IV. . . . Also, most preparations are obtained from the digestion of casein or fibrin, and hence contain a high percentage of glutomic acid—known to be fine "brain-food," but also known to cause emesis when given IV. . . . All the evidence suggests that oral administration is preferable.

About fifteen months ago a case-analysis was published which dealt with the BITES AND STINGS of small desert "animals." The question arose at that time whether bites and stings could be modified by anti-histamine drugs. . . . Correspondence with two drug manufacturers resulted in the statement that any remarkable effect was doubtful, even in insect bites, though very few data had been reported. . . . Now, in a single week, abstracts are published which show that the anti-histaminics (1) relieve the pruritis of mosquito bites in both sensitized and non sensitized people; (2) reduce the reaction of tsetse fly bites; and (3) relieve the pain of bee stings almost at once, if given soon after the bite.

The management of ACUTE CHOLECYSTITIS is a tough problem. A recent tendency, in skilled hands, is to operate as soon as the patient can be evaluated and prepared. . . . Cholecystectomy is said to be safe. Cholecystostomy may be done, with clearance of the cystic duct, if there is jaundice or severe inflammation; the gall bladder is then removed about six months later. . . . CHRONIC CHOLELITHIASIS may be found in 10 to 20 per cent of adults over 30 years of age. About 50 per cent of such cases are symptomatic. Surgery may be done in the symptomatic type during a free interval.

The prescription of SEDENTARY HOBBIES for individuals with heart disease is a necessary part of therapy. The type of hobby need not be determined by the patient's age. It should be constructive. . . . Furthermore, and because of their tendency to tension and coronary lesions, it has been suggested that physicians should adopt such hobbies early in life!

Considering the number of bacteria which are present in this world, it hardly seems possible that warnings against PENICILLIN-RESISTANT STRAINS could be of importance. Yet they are. . . . Previous to 1944, resistant strains were rare. In 1944 Spink found 12 per cent of 68 strains of staphlococci to be resistant. In 1944 Barber et al found 14 per cent of the strains seen in her hospital to be resistant; and in 1947 the percentage was 38; and in 1948 it had risen to 59%. . . . Bacteria other than staphlococci have not been so thoroughly reported, but several types show similar changes. . . . Nichols and Needham of the Mayo Clinic have successfully treated cases in which there were penicillin-resistant staph. aureus infections with aureomycin. The drug was given intravenously to avoid the nausea caused by oral administration. A resistance to aureomycin is not common, nor great.

Caffeine has long been used to reinforce the effect of certain other drugs. It has now been found that 100 mgm. of CAFFEINE plus 1 mgm. of ERGOTOMINE TARTRATE (E. C. 110—San-

doz) is more effective than ergotamine tartrate alone for migraine, histamine headache, and (to a lesser extent) for tension headache.

Facilities for the help of sub-normal individuals sometimes exist with scant publicity. For example THE DEAF AND THE HARD OF HEARING in Washington, D. C., have the following special associations available,—5 public schools, 2 private schools, 9 teachers, 9 hospital services, 2 schools for training of teachers, 3 organizations for help, 5 literary and social societies, 8 religious groups, 2 athletic clubs, 3 theaters with hearing-aids, 5 federal or municipal services, and a newspaper.

Some of the antibiotics have failed to be as effective for SKIN INFECTIONS as they are for internal lesions. One of those which can not be used parenterally (BACITRACIN) is apparently very valuable when applied locally. . . . Eichenlaub and Olivo of Georgetown have confirmed the reports of Meleney and others that pyogenic dermatitis lesions are usually improved or cured in 1 to 2 weeks. . . . In their series of 50 cases, 44 responded. Only two showed sensitivity to the drug. An ointment of 500 U. per gram is used. Boric acid may be used in conjunction, since bacitracin is inactivated by hydrogen peroxide and potassium permanganate.

The decrease in the incidence of thromboembolic phenomena (and in mortality) following the use of anticoagulants in cases of ACUTE MYOCARDIAL INFARCTION is notable. . . . Heparin needs no "control" tests, but must be given hypodermically to a sick patient every four hours; it can therefore be best used at the outset, and until dicumarol takes effect. . . . Dicumarol is given by mouth, but for real safety a prothrombin time should be done daily. . . . In one series of 100 cases, bleeding occurred only once. Other methods of treatment should be used concurrently.

One might follow, or even precede, a paragraph on ANTICOAGULANTS by a note on their antagonists. . . . Dicumarol can usually be counteracted by transfusions and parenteral Vitamin K. . . . Heparin effect is eliminated by the protamines, toluidine blue, and a few hours of time.

A Mayo Clinic report on the results of surgery for 640 cases of DUODENAL ULCER revealed a mortality of only 3%. Half of those who died had pre-operative gastric retention. . . . Vagotomy is on the wane in their hands. Subtotal gastrectomy is the method of choice.

The simple bilateral "massage" test for a SENSITIVE CAROTID SINUS should be a part of every complete physical examination, especially when symptoms of syncope, hypotension, bradycardia, etc., are present. . . . The types originally described by Weiss and Baker were those with cardiac inhibition, vaso-depression or cerebral effects; but they may be combined. . . . Discovery of "tight-collar" cases, or the accidental production of a syndrome while palpating the thyroid gland, can be a very dramatic diagnosis.

To most of us KETOSTEROIDS are something the biochemists have slipped over on us since medical school days. Steroids are now known to be present in plant and animal sterols, bile acids, cardiac glucosides, sapo-genins, sex hormones, and sterols from the adrenal cortex. . . They all come from a basic formula—the 4-ring nucleus. They are called "17-ketosteroids" because "17" is the position of substitution on the nucleus (except in sex hormones). . . . The value of ketosteroids in diagnosis depends upon their amount, proportions, and type, as found in the urine. They help to diagnose disease or dysfunction of the adrenals, pituitary, thyroid, and gonads. . . . From here on, all one needs to remember is where to look up the latest data on the subject.

The usage of FACIAL PROSTHESES, chiefly for the nose and ears, has been given a lift by a clever trick in casting. . . . Because the spare part deteriorates after a few months, a master mould of metal or plaster is kept on hand, and replacements may be made at intervale, PRN. . . The ideal prosthetic substance has not yet been found, but pliable plastics and prevulcanized latex are being tried.

Note No. 3 on BARBITURATES,—Restricting the prescription and sale of sleeping pills will not prevent their abuse. Adulteration by an emetic is far from being an accepted procedure. Publicity about the hazards of the drug has not decreased suicides, which now total about 1,000 per year. . . . We must be prepared to treat as well as prevent. Therapy of the patient must be preceded by diagnosis, which depends on positive evidence as well as ruling out the "vowel" causes of unconsciousness, — (alcohol, epilepsy, injury, opium, uremia, plus diabetic conditions, etc.). . . . Immediate methods consist of eliminating the ingested drug and counteracting the effects of what has been absorbed. The sequence depends on the depth of unconsciousness,—if the patient has reflexes and is responsive, gastric lavage, and a purge come first; if unconsciousness is deep, start off with the antagonists. . . . The choice of picrotoxin of nikethamide seems to be in favor of the latter. Watts and Ruthberg of N. Y. C. found, in a series of 99 cases, that nikethamide was superior in effect, and safer. Convulsions occurred with picrotoxin, but not with nikethamide. It is easier on the patient, and allows nurses to use it. . . . Five to 10 c.c. of a

25 per cent solution of nikethamide is given intravenously, followed by 5 c.c. every 5 minutes for an hour. A basal dose of 5 c.c. per hour is also given by IV drip. Booster doses were later given every half to one hour, depending on the severity of poisoning. . . . Initial therapy must be continued until the patient responds to questions, rather than a return of reflexes.

This column can now claim a journalistic career of ONE YEAR'S REGULAR PUBLICATION. It seems an apt time to look at the record. . . . The symbols in the TITLE of the column, incidentally, mean "Therapy, Diagnosis, and Physicians." . . . The OBJECTIVES, as stated in the first collection of paragraphs, have generally been met,—"topical news on medical subjects which may have special interest to Arizona physicians." . . . There have been a few scoops, a few timely summaries, and a couple of dozen topics which have had to be discarded as passe. There have been a few flies in the happy ointment,—"darvisul" has not yet been found to wipe out poliomyelitis; "methadon" has disadvantages which the early though reputable reports did not list; etc. . . . We will be more alert, more diligent, and more careful in the future—or at least we will put obvious labels on discoveries which are still in the "possible" stage.

PHOENIX CLINICAL CLUB

March 14, 1949
Massachusetts General Hospital,
Case Record No. 33251

A twenty-three-year-old single woman entered the hospital complaining of blueness of the hands, face and feet.

No abnormality at birth or in development as a child had been noted, but her mother stated that as a child she had not played with other children very much and had always been "lazy." At the age of twelve a mild, dull pain in the chest, located to the left of the lower portion of the sternum, had been noticed. At that time a bluish discoloration of the face and hands was first observed. There were no associated symptoms that seriously incapacitated the patient, although she noticed that if she exercised as other children she had headaches and became blue and somewhat weak. These symptoms, however, did not prevent her from roller skating and dancing. The pain disappeared promptly, but occasionally thereafter she was aware of a slight, dull pain in the chest, without particular relation to any activity. In addition, she complained that the eyes frequently became "bloodshot" and that the cold aggravated the blueness of the extremities. A gradual clubbing of the fingers from that time was noted. Three years before admission the patient went to a physician with these complaints. She was told that she had low blood pressure and was given some "pills" which she took without benefit; she did not, however, consider the symptoms severe enough to return. A year later she saw a dentist for bleeding gums and was referred to her physician for a blood examination, which was negative. Fourteen months before entry she attempted to join the WAVES and was told that everything was normal except for blueness, clubbing of the fingers and an abnormal chest film. She was referred by the Navy to a sanatorium, where extensive studies for tuberculosis were negative. At the time of admission the patient complained only of cyanosis and clubbing of the fingers and toes. She felt perfectly well, being able to exercise strenuously without ill effect other than an increase in cyanosis, which she thought had gradually become more prominent.

The past history revealed no serious illness. The patient had had mumps, measles and chicken pox as a child. She had had occasional sinusitis and a chronic morning cough productive of a teaspoonful of sputum. The gums had always bled easily. There was no history of familial disease.

Physical examination on admission revealed a well developed and well nourished girl in no distress. There was a striking cyanosis of the face, hands and feet with severe clubbing of the fingers and toes. The conjunctivas were markedly congested, and examination of the fundi showed extremely dilated veins with tortuous arterioles. The throat was a dusky red. There were no abnormal pulsations in the neck. The chest was clear. The heart was normal in size. The pulmonic second sound was louder than the aortic second sound. No murmurs were heard. One examiner described an inconstant pulmonary systolic murmur. The liver and spleen were not palpable. The extremities showed good pulsations. Neurologic examination revealed a right knee jerk that was less active than the left.

The temperature was 99.6°F.; the pulse 80; and the respirations 15. The blood pressure was 100 systolic, 80 diastolic, in the right arm and 95 systolic, 78 diastolic, in the left.

Examinations of the blood showed red-cell counts ranging from 7,100,000 to 8,790,000, with hemoglobins of 21.5 to 24.9 gm., and a white-cell count of 12,000, with a normal differential. The venous blood had an oxygen content of 16.7 and an oxygen capacity of 28.5 vol. per cent, with a

hematocrit of 69.7 per cent. The arterial blood had an oxygen content of 24.85 and an oxygen capacity of 27.7 vol. per cent, with a hematocrit of 67 per cent. The oxygen saturation was 89.2 vol. per cent. The circulation time with ether was 11 seconds and with Decholin, 23 seconds. The vital capacity was 3100 cc. Urinalysis was normal. An electrocardiographic tracing was normal.

X-ray examination of the chest revealed an extensive "flecky" area of increased density in the right middle lobe, mainly in the anteromedial segment. There was also a fine network of increased density in the left lung field laterally that was thought to be in the lingular portion of the left upper lobe. Both leaves of the diaphragm were well defined and showed good motion. The hilar shadows were normal. The heart was normal in size, shape and action. Fluoroscopy showed evidence of pulsations of the major branches of the right pulmonary artery somewhat farther away from the hilus than usual. The finer branches showed no abnormal pulsations. The hands and feet failed to show any evidence of pulmonary osteoarthropathy.

The patient's condition remained unchanged. On the sixteenth hospital day an operation was performed.

DISCUSSION

Dr. Robert S. Flinn:

This 23-year-old girl developed a bluish discoloration of her face and hands at the age of 12, followed by a gradual clubbing of her fingers, blueness of the extremities and frequent bloodshot eyes. On examination her heart was found to be normal in size and in configuration with normal heart sounds. She had polycythemia, evidence of deficient oxygen saturation and an area of increased density in her right middle lobe. That, as the English say, is the guts of the situation. Let us turn for a moment to a discussion of the laboratory findings. The circulation time with ether is given as 11 seconds. This represents the time elapsing between the injection of a few minims of ether into an antecubital vein and its arrival at the pulmonary capillaries where it is detected by a slight cough. The normal arm to lung circulation time for ether is 6 to 10 seconds with an average of 8 seconds. This is an index of functional activity of the right heart. Chronic pulmonary disease does not usually affect the test. In the case under discussion since the circulation time was within 1 second of what is considered normal, I do not believe we can place any interpretation upon it. However, the arm to tongue time with decholin is given at 23 seconds. The normal readings vary between 10 to 16 seconds with an average of 13 seconds. The difference between the arm to tongue and the arm to lung time marks the lung to tongue time. It varies under normal conditions from 4½ to 10 seconds. In our case, it is 12 seconds. Prolongation of the lung to tongue time, that is, with the arm to lung time normal, is occasionally observed in left heart failure. I do not believe that we can place much significance on this test in the case under discussion except to point out that in polycythemia there is frequently some delay in the blood flow.

The oxygen saturation was 89.2 volumes per cent. Under normal conditions the blood which passes through the lungs is fully or nearly fully oxygenated. The blood which is returned by the pulmonary veins to the left auricle is 94 to 97 per cent saturated with oxygen. The fact that arterial blood is not 100 per cent saturated has long been believed to be due to the fact that the bronchial veins pour some venous blood into the pulmonary veins. However, recent experimental evidence indicates that the slight oxygen unsaturation may be due mainly to a technical error. As the blood circulates through the capillaries, approximately 22 per cent of the oxygen in the arterial blood is given off to the tissues. It follows that the oxygen saturation of normal venous blood is 72 to 75 per cent. Thus normal arterial blood contains 94 to 97 per cent oxyhemoglobin and 6 to 3 per cent reduced hemoglobin, and normal venous blood contains 72 to 75 per cent oxyhemoglobin and 28 to 25 per cent reduced hemoglobin.

Inasmuch as one cubic centimeter of oxygen is taken up by 0.75 gram of hemoglobin, one gram of hemoglobin takes up 1.33 c.c. of oxygen, and 15 grams of hemoglobin will carry 20 c.c. of oxygen. It follows that blood with a normal hemoglobin of 15 grams per 100 c.c. will carry 20 volumes per cent of oxygen; in other words, the oxygen capacity of normal blood with 15 grams of available hemoglobin per 100 c.c. of circulating blood is 20 volumes per cent. Since the oxygen saturation of the arterial blood is 94 to 97 per cent, its oxygen content is approximately 19 volumes per cent. Inasmuch as 22 per cent of the oxygen in the arterial blood or approximately 5 volumes per cent is given off in the tissues, normal venous blood has an oxygen content of 14 to 15 volumes per cent and 5 to 6 volumes per cent of reduced hemoglobin.

So it can be seen that in order to determine the oxygen capacity and the oxygen content it is

necessary to know the amount of hemoglobin present in the blood sample to be examined. Since the hemoglobin varies from 21.5 to 24.9 grams, a difference of almost 4 grams, I am not attempting to work out what would be the normal oxygen capacity for this patient. We do know that this patient had visible cyanosis and that there must be at least 5 grams reduced hemoglobin per 100 c.c. of circulating blood to produce it. Consequently one-third of the blood circulating through the capillaries must be in the form of reduced hemoglobin to cause cyanosis.

Let us discuss for a moment the probable causes for this arterial oxygen unsaturation. 1. It is due to a congenital heart lesion; 2. a generalized pulmonary condition such as Ayerza's disease in which none of the blood leaving the lungs is properly aerated, or 3. to a local lung condition allowing by some means such as a shunt, the passage of a proportion of the blood directly from the pulmonary artery to the pulmonary vein. Since no abnormality at birth or in the development of the child was noticed, since the heart is normal in size and in configuration, and since no murmurs were heard, I think that one cannot possibly entertain a diagnosis of any congenital heart lesion. Since the x-ray of the chest showed no suggestion of any generalized pulmonary fibrosis, I do not see how we can consider Ayerza's disease as a possible factor. However, since the x-ray examination did show a definite shadow in the antero-medial segment of the right middle lobe, it seems probable that there may be some abnormal arteriovenous communication in the lung in the nature of a cavernous hemangioma.

Hepburn of Toronto, in 1943 in reporting one of the first cases in which successful removal of the hemangioma was carried out, stated that it is a rare condition and had not been encountered previously at the pathological department at the University of Toronto. Beierwoltes and Biron in 1947, to seven previously reported cases added another case of pulmonary arteriovenous aneurysm with secondary polycythemia which was successfully operated upon. As in the other cases, the oxygen saturation quickly rose to normal following operation with reduction in the hematocrit reading. These authors state that the most important pre-requisite for making a diagnosis is the awareness of this lesion as a cause of a cyanosis and polycythemia. The patient may be of either sex and is usually between 20 and 30, with cyanosis which has recently become worse. Symptoms of anoxia predominate with hemorrhage from the superficial hemangiomas usually from the nose. Cyanosis has often been noticed in siblings and parents. Only three patients had murmurs produced by the vascular communication.

In the Journal of Thoracic Surgery February, 1948, Maier, Himmelstein and Riley report a case of arteriovenous fistula of the lungs with superimposed bacterial endocarditis successfully treated with penicillin followed by surgical removal of the involved portion of the lung. In 1946 Lindgren of Stockholm reports three cases of arteriovenous aneurysm which were verified at operation. He points out that the classic pathological anatomical change is a distended afferent artery and a distended efferent vein. Between the arterial and venous systems there is either a direct communication to one or several of the larger vessels or a tangle of more or less distended veins instead of capillaries. Since the arterial pressure is transmitted directly into the malformed vessels and into the veins, these become increasingly dilated and sometimes rupture. He points out that it is usually easy to make a correct diagnosis of pulmonary arteriovenous aneurysm by ordinary x-ray examination. The characteristic feature is the broad band-shaped densities connected with the hilar vessels and in the periphery communicating one with another either through vascular formations or rounded formations resembling tumors. Valsalva's maneuver causes the densities to shrink.

In the Annals of Internal Medicine for Nov. 1948, Captain John H. Moyer of Fort Sam Houston, reported two cases of hereditary hemorrhagic telangectasis associated with pulmonary arteriovenous fistula in which a cure of one of the cases was obtained after a pneumonectomy. They point out the clinical manifestations seem to be extremely variable. Small fistulae cause no complaints while large ones give rise to a complex of symptoms and signs which almost characteristically occurred in the 15 cases previously described in the literature. Arteriovenous cysts of the lung may become fairly large before the typical trial of cyanosis polycythemia and clubbing of the fingers develops. According to some observations, approximately 30% of the blood must be shunted before cyanosis become manifest in otherwise normal individuals. The effects

of the anoxemia due to the pulmonary shunt and the compensatory mechanism are interesting from a clinical and laboratory point of view. A compensatory change consists of an increase of blood volume affecting the cell volume rather than the plasma volume, increase hemoglobin concentration and an increase of red cells closely paralleling the changes seen in the relative anoxia of subjects living at high altitude for long periods of time. In both conditions, these changes are proportional to the degree of unsaturation of the arterial blood.

The lack of cardiac enlargement is significant. An enlarged heart was observed in only two cases, one of which had mitral stenosis and the other case the enlargement was due to congestive failure secondary to chronic myocardial disease. Kennedy and Burwell have noted that in chronic peripheral arteriovenous fistulae there is an increase in the cardiac output, blood volume and venous oxygen tension near the fistula and mild to moderate cardiac hypertrophy. The lack of cardiac hypertrophy in cases of chronic pulmonary arteriovenous fistulae has been attributed to the low pressure in the pulmonary circulation.

TREATMENT

A small fistulae usually asymptomatic may require no treatment at all. However, it is advisable to observe these patients over long periods of time since these fistulae may increase in size —while others may develop. Symptomatic arteriovenous fistulae necessitates surgical intervention. The danger, of course, in the non-operative ones are venous thrombosis because of the marked polycythemia and the increased blood viscosity.

To return to the case under discussion, we have in summary, then, a 23-year-old patient who has cyanosis, polycythemia, clubbing of the fingers and a characteristic x-ray shadow in the chest. A diagnosis of congenital heart disease is untenable as is also one of Ayerza's disease. By exclusion, then, a diagnosis of arteriovenous fistula can be made.

Differential Diagnosis

Dr. Earle M. Chapman: A pulmonary murmur was heard. Does that mean that it was heard over the lung or in the second left interspace? That is an important observation, and it needs clarification.

Dr. Ronald C. Sniffen: The record is not clear on that point.

Dr. Chapman: May we see the x-ray films?

Dr. Stanley M. Wyman: The film of the chest shows mottled density lying in the anterior medial segment of the right middle lobe as seen in the anteroposterior projection. It is seen in the lateral view lying in this triangle. There was said to be fine mottling in the lingula of the left upper lobe. I cannot be sure of that finding. I think it should be noted that there is a rounded, sharply defined shadow extending outward and downward at this point, coming down to this region, which suggests the pulmonary artery. The lateral view of the skull shows no definite abnormality.

Dr. Chapman: May I ask if pneumothorax was done and if a further definition of the mass was obtained?

Dr. Sniffen: Pneumothorax was not done.

Dr. Chapman: This case represents a rare clinical syndrome that would not have been diagnosed or treated ten years ago. Indeed, its diagnosis was missed on three occasions during the patient's life. The characteristics are so clearly set forth in this history that I have no hesitancy in making a diagnosis of an anteriovenous fistula of the lung, with secondary polycythemia.

Only two other conditions have to be considered in the differential diagnosis, and they are congenital heart disease and polycythemia vera. The latter is excluded by the failure to find immature white cells or basophilia in the smear. Congenital heart disease is practically excluded by the entirely normal cardiac findings.

This rare disorder is often congenital but possibly traumatic in origin and yet the few cases reported in the literature since 1938 seem to have established a group of symptoms and signs that should indicate the correct diagnosis. These are cyanosis, clubbing of the fingers and toes, and symptomatic polycythemia usually in a young person with a normal heart and an obscure pulmonary lesion by x-ray study. Some patients have a continuous murmur over the lesion if it is the cavernous type connecting with the pulmonary artery.

The hazards of continuing with the lesion are that it may rupture and lead to sudden death in pulmonary hemorrhage, such as in the case described by Rodes in 1938,[1] which at autopsy showed three hemangiomas in the right lung and one in the left lung; or the patient may be in-

capacitated by the symptoms of anoxemia. Such were the symptoms that led to the first successful removal of the right lung containing such a hemangioma in 1942.

Since then several cases have been successfully operated on, and the patients have lost the cyanosis immediately after operation, the blood counts have returned to normal, and the symptoms have disappeared. Disappearance of the symptoms, however, depends on the removal of the entire lesion and the absence of multiple arteriovenous fistulas.

There are some interesting data and clinical points in this case that I think deserve some comment. For instance the bleeding of the gums and suffusion of the eyes and mouth are clinical signs consistent with a polycythemia, and yet they seem to have been overlooked as diagnostic signs. The pulse rate was slow, and this suggests that the cardiac output was normal, since according to Marey's law a rapid pulse can be due to a low blood pressure and not to an increased venous pressure.

Having seen the x-ray film I place the lesion in the right lung rather than the left, although it is possible for such a lesion to exist on the left side as well.

Fluoroscopy showed, "evidence of pulsations of the major branches of the right pulmonary artery . . . . " That is why I was concerned with the so-called "pulmonary murmur" and with the decision whether it was a murmur over the heart. The history stated that no heart murmurs were heard and later that a "pulmonary murmur" was heard. To me, that means a murmur over the lungs. The sign has been described in some cases of arteriovenous fistula of the lung.

The oxygen saturation in itself could be a significant diagnostic procedure. The patient had a slightly slow circulation time; with an arteriovenous lung fistula or even in pulmonary congestion one could expect even greater delay in the blood circuit through the lung.

In conclusion, then, I believe that this case is characteristic possibly of a cavernous hemangioma type of arteriovenous fistula of the lung, and I assume that at operation perhaps all or part of the right lung was removed. It would be interesting to hear from the others about the subsequent course.

Dr. Sniffen: Dr. King, have you any comment?

Dr. Donald S. King: The patient came to the sanatorium with a diagnosis of questionable tuberculosis and was sent to this hospital from there.

Dr. John T. Quinby: I believe that I was the first to see the patient in our Out-Patient Department with some of the fourth year men, and I thought that she had congenital heart disease. I followed her after entry into the hospital. Preoperatively the hematocrit was 67 per cent and the oxygen saturation 89 vol. %.

Dr. Sniffen: Is such cyanosis usual with a not very low oxygen saturation?

Dr. Quinby: As I understand it, the cyanosis depends on concentration of the reduced hemoglobin. With a high total hemoglobin, an unsaturation of 6 or 7 per cent apparently leaves enough reduced hemoglobin to cause a dusky color. Another interesting problem is that the cyanosis and exertion limitation were progressive as the patient grew older, suggesting the magnitude of the lesion was increasing.

Dr. Chapman: I intended to comment on the presence of cyanosis. The oxygen saturation was low,—the normal figure should not be under 95 vol. per cent, and in this case it was 89, representing slight diminution,—but this was compensated for by the polycythemia. In most patients with congenital heart disease when one third of the blood is shunted through from the venous to the arterial side, the reduced hemoglobin goes up from 1 to 4 vol. per cent, and cyanosis becomes established. It is unfortunate that the volumes per cent of reduced hemoglobin were not available.

Clinical Diagnosis

Hemangioma of the lung.

Dr. Chapman's Diagnosis

Arteriovenous fistula of lung, with secondary polycythemia.

Anatomical Diagnosis

Cavernous hemangioma of right middle lobe of lung.

Pathological Discussion

Dr. Richard H. Sweet: As Dr. Chapman has said, this is a characteristic entity. The correct diagnosis was made ahead of time in this case by the Medical Department and concurred in by the surgeons. At that time there had been only four or five such cases operated on successfully, at least according to reports in the literature. All of them, I believe, had been pneumonectomies with the exception of one patient of Dr.

Janes in Toronto. This case, which was bilateral, was successfully operated on in two stages. The case under discussion was a so-called "hemangioma," which I prefer to call an arteriovenous fistula. There was one enormous pulmonary artery, probably as large as the whole pulmonary artery should be normally, and there was a corresponding enormous vein, which led out from the middle lobe. In the middle lobe there were numerous dilated vascular channels. So far as I know this is the first case treated by lobectomy alone. Since then, by the way, we have had a similar case—a lesion in the middle lobe—in a child two years of age, who presented the same clinical picture and was cured by surgery. On visiting Dr. Alexander's Clinic in Ann Arbor I recently discovered there was a third such case with arteriovenous fistula in the middle lobe. It had been thought to be the first one that had been resected because we had not published our two cases.

Dr. Sniffen: After removal the external surface of the right middle lobe showed a number of small blebs without pleural reaction. Three large vessels entered the lung at the hilus. One of these vessels followed the course of the anteromedial bronchus, and 2 cm. beyond its origin it was broken up into a honeycomb of large anastomosing, vascular channels that occupied an area 9 by 4 by 3.5 cm. in the medial portion of the lobe and accounted for the blebs on the external surface. The lesion was not encapsulated but showed no evidence of growth. The channels were lined with endothelium, with localized points of subendothelial fibrous thickening.

The postoperative findings are of some interest. Eight days after operation the hematocrit had fallen to 62%, and the oxygen saturation was up to normal. The hemoglobin remained elevated. In the few cases I have read about, such as the patient studied by Hepburn and Dauphinee[2] the blood vessels, including the hemoglobin, had returned to normal in eight days. In the case under discussion about six months elapsed before the hemoglobin and red-cell count fell to normal figures.

Dr. Sweet: Several months after operation the patient showed some reduction in the clubbing of the fingers, and she was no longer cyanotic. I think that one comment is of interest regarding Dr. Sniffen's remark about the rate at which the hemoglobin returns to a normal level. Until this operation was done,—I have not followed the reports in the literature since then,—all the patients operated on suffered alarmingly of exsanguinating hemorrhages on the operating table. I, therefore, assume that the rapid reduction was due to blood loss. We are sensitive to that, and this patient did not suffer any appreciable loss of blood during the operation. I think that possibly this was responsible for the slow return of the hemoglobin to the normal level.

Dr. Howard Sprague: Certainly, in peripheral arteriovenous communication, one would expect enlargement of the heart because of the increased flow. We have to agree possibly to an increase in pulmonary venous pressure. Does it mean that the right ventricle can stand increased circulation better than the left ventricle? There are so few of these cases that we have not been able to draw a conclusion, but I think that it is of considerable interest that this patient had an increase in flow.

Dr. Sniffen: Another point in reference to Dr. Chapman's remarks: it is stated in the record that the blood examination two years before admission was normal in this case. In Hepburn's case the patient had been followed carefully in a sanatorium and at that time she had a normal red-cell count and hemoglobin. Only in the last year and a half did the red cells and the hemoglobin increase.

Dr. Sweet: How about cyanosis?

Dr. Sniffen: It was present.

Dr. Sweet: When the blood counts were normal?

Dr. Sniffen: Yes, presumably because of the arteriovenous shunt and before secondary polycythemia had become established.

Dr. Chapman: I should suggest that the lesion was expanding in type and becoming enlarged, and that the degree of polycythemia was in relation to the size of the shunt. In this case, as the tissues became distended, there was more bleeding. It is an interesting point that Dr. Sprague has made, and one that has been made in other cases. It is surprising how the heart can accommodate itself to this extra load.

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1 Rodes, C. B. Cavernous Hemangioma of Lung with Secondary Polycythemia. J.A.M.A. 110: 1914. 1938.

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STOP DESPOTISM WITHIN OUR RANKS!

The time has come to call a halt to compulsory attendance at meetings. For years we have railed, rightfully, against proposed medical compulsion of various kinds by government. Let's clean our own house, now!

Institutions, some of them not at all democratically controlled, have set standards for medical, surgical and hospital practices which work real hardship by forcing physicians to attend a very high percentage of meetings or lose their right to use their workshops. We admit these rulings have some advantages in disciplinary power and encouragement of improved scientific endeavor in presenting programs. But disadvantages far outweigh advantages.

Why should limited specialists be literally forced to spend whole evenings listening to long reports by limited specialists in other fields—material which can in no way improve their own knowledge and practice? The same length of time spent upon general subjects or material in their own fields in their own libraries—or even just relaxing apart from science—would make them better doctors.

Small communities with only one or two hospitals do not pose the critical problem that now plagues doctors in larger centers. Most of us in cities are now forced to spread ourselves entirely too thin. We are obliged to attend staff meetings of several hospitals, patronize specialty groups, support the committee activities of all of them. There are no evenings left for one's family, none for simple living and being a human being. One feels like crying, "I can't take any more; another meeting this week will spell the last of me!"

Then the County Medical Society suffers. Here is one meeting we are not compelled to attend, yet it is the organization that is the foundation of all medical organizations and its meeting is the one we all should attend. This one depends upon its merit to attract our attendance—not on someone checking off names to delineate good boys from bad boys. The County Society is the hub of all local medical activity. It not only advances our science; it formulates medicine's public policies and develops educational pursuits for laymen who need it most of all; it directs opposition to destructive legislation; it relegates opponents of scientific medicine to their proper place; it uncovers and exposes subversive elements. Without actively and enthusiastically supported county societies our public service efforts will lag, public relations programs will deteriorate into mere efforts to obtain publicity, lay educational enterprises will fail, adverse legislation and unscientific cults will prosper, and regional and national parent medical organization will suffer in proportion.

Our county medical societies really need us and we really need them, but there are only seven nights in a week and human endurance has a definite ceiling which becomes lower during and after middle age. This physiological fact cuts down too many doctors in their most productive and useful years.

Yet we must earn our living, so our staff positions must be preserved. We cannot risk "being relieved" of hard-earned places in our workshops. So we have to be officially "excused" for non-attendance. We are disciplined like grade school children.

Are we not mature enough to choose those meetings to which we may contribute or from which we may derive benefit? Are we not loyal enough to attend those whose actions and recommendations control the honor, dignity, and future of medicine? If so, let us be heard by those who have carried so-called standardization to the level of despotism.

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ARIZONA'S BLUE CROSS

One of the finest examples of cooperation between the Blue Cross Board of Directors, Blue Cross member hospitals and the public as represented in the Blue Cross membership was found recently in a series of meetings held to explain to representatives of member groups the basic Blue Cross philosophy, the place of Blue Cross in the economy of hospitals, the financial problems faced by hospitals because of rising costs, and the resulting necessity for increasing the Blue Cross membership dues.

First of the three meetings was held at Good Samaritan Hospital, Phoenix, with Dr. Preston T. Brown, Phoenix, Vice President of Arizona

Blue Cross, presiding. Charles Korrick, former Blue Cross President and a Member of the Board of Directors, outlined the history, aims and purposes of the organization. Mr. Korrick incidentally also is an employer who pays the full Blue Cross costs for his employees, and is a member of the Board of Directors of St. Monica's Hospital. Guy Hanner, Good Samaritan Hospital Administrator and Secretary of the Arizona Hospital Association, spoke of the hospitals problems, and L. Donald Lau, Blue Cross Executive Director, explained the fundamental differences between the service benefits of Blue Cross and the limited dollar indemnities of commercial insurance companies which offer hospital insurance plans.

A second meeting was held at St. Monica's Hospital, Phoenix, with Dr. Brown, Mr. Korrick and Mr. Lau again presenting their views on the respective phases of the situation, and with Emmett McLoughlin, Superintendent of St. Monica's—and a member of the Blue Cross Board of Directors—speaking for the hospitals.

The third meeting was held at the Tucson Medical Center, with Charles D. McCarty, Blue Cross Board Member; the Rev. George Ferguson, President of the Board of Directors of the Medical Center, and Mr. Lau speaking, and with Ned F. Parish, Assistant Director of Arizona Blue Cross, presiding.

Most of the large groups of Blue Cross members in the Phoenix and Tucson areas were represented at the meetings and discussion was free and constructive. General approval of the action of the Blue Cross Board in raising the membership dues rather than reducing the service benefits was voiced at all three meetings.

Blue Cross Member Hospitals already have been notified directly by Mr. Lau (in a memorandum dated June 21st) of the dues increase effective August 1st, and of the formula for allocating the increased income to Member Hospitals on all Blue Cross hospital admissions on and after October 1st.

It will now be more important than ever for Member Hospitals to report immediately the admissions of all Blue Cross patients. The Blue Cross office cannot keep before it an accurate, continuous picture of its financial condition unless this is done. For example, the boys with the slide rules were thrown a terrific curve early in the year when, during March, some 300 hospital cases for which no admission notices had been received were submitted for payment by the hospitals. These cases were admitted during January and February. To say that the financial picture was distorted, is putting it mildly. Closer attention to the prompt reporting of admissions—which incidentally is required under Section 5 of Member Hospital contracts—will be greatly and especially appreciated in the future.

The welcome mat is out for Dr. Martin F. Heidgen, who has succeeded Clyde Fox as Administrator of the Tucson Medical Center. Mr. Fox, who was President of the Arizona Hospital Association, resigned effective June 15th to become Administrator of the Washoe General Hospital at Reno, Nevada.

Dr. Heidgen has served as Superintendent of the Memorial Hospital of DuPage County, Elmhurst, Ill., in the Chicago Blue Cross Plan area, for the past seventeen years. A native of Milwaukee, Wis., he obtained his bachelors' degree from Marquette University at Milwaukee, and his M. D. degree from the University of Chicago. He is a member of the editorial advisory board of HOSPITAL MANAGEMENT, national publication dealing with hospital administration, and is a fellow of the American Medical Association. He also is a member of the Illinois Hospital Association and the Chicago Hospital Council.

Every nine seconds during 1948 a Blue Cross member had a hospital bill paid. National Blue Cross membership (including Canada and Porto Rico) passed 33,000,000 at the end of March. Arizona had a little more than 106,000 members at the end of May.

May we correct what is apparently a wrong impression? Since the changes in our contract last February 15th, it is apparent that some hospitals have misunderstood the new procedures in regard to payments for out-patient care. The contract states: "In emergency room of Member Hospital, an allowance of up to $8 per admission will be provided to a subscriber." This means that the per diem cost of your hospital for out-patient care is to apply for all charges up to $8. In the event the charge exceeds this amount, the hospital can then collect from the patient the difference between the charge and the allowance of $8. This action was taken by the Hospital Committee of Blue Cross and approved by the Board of Directors to permit the

hospitals to receive more adequate compensation for expensive out-patient care.

Are you writing to your Congressman? If you are, here are some tips on how to do it properly, offered by the Chamber of Commerce of the United States: (1) Address him as The Honorable John Doe, M. C. (for Member of Congress) or U. S. S. (for U. S. Senator)—and be sure of who is what. (2) Be Local; Tell him how a National question affects your business, your industry, your community. (3) Be Business-like; brief but not terse. (4) Be Specific; if you're for something, say so. If not, don't hedge. (5) Be Polite; Congressmen deserve dignified treatment. (6) Be Reasonable; seek only possible things. (7) Be Yourself; use your own letterhead and letter style. 8) Request Action; your man is elected to do something. (9) Ask For An Answer; you've told him where you stand, now ask him where he stands. (10) Be Appreciative; thank him for good votes, compliment his better speeches, and praise his staff, too.

Those tips are worth pasting in the bonnet in these days, when so much legislation affecting the whole of the health picture in this country is before the houses of Congress.

And speaking of National Legislation, here's a gem from testimony of Dr. Paul Hawley, Chief Executive Officer for Blue Cross and Blue Shield, before the Senate Committee on Labor and Welfare on May 31st: "Health is a most important national asset. But it is by no means the only important asset and it is closely related to others. If through expenditures upon health, a crippling burden is placed upon the budget of the Government, the stated objective of S. 1679 (the Administration's compulsory health insurance program) will be defeated. The best medical care in the world cannot maintain good health if handicapped by a general depression arising out of confiscatory taxation."

Tempe Clinic-Hospital observed American Hospital Day on May 12th with a luncheon and reception at which Mrs. Pohle, wife of Dr. Ernest Pohle, Superintendent of the hospital, outlined the obligations of a hospital to its community, and a talk was given by the Rev. Dr. Staley of the College of Medical Evangelists, Los Angeles. Dr. Staley, representing an organization which operates in about eight countries, decried the trend toward socialized medicine and recommended support of Blue Cross and Blue Shield as important bulwarks between the American public and the Welfare State. Guests were taken on a tour of the hospital as the final item on the hospital day program.

Statistically speaking, payments to hospitals by Arizona Blue Cross for the first half of 1949 totalled $426,668.27. Patients received care for a total of 38,804 patient days. Of this last total, 835 were out-patient days. The average hospital stay for Blue Cross members (bed-patients only are included in this figure) was 6.18 days per case. This is a little up from the 1948 average of 6.13 days per hospital case for Blue Cross members.

We think the new News Service of the American Hospital Association is one of the finest information sheets in any field, and undoubtedly you will be seeing excerpts from it in coming bulletins of the Arizona Hospital Association. Its material is written to be passed along to local and state hospitals.

Blue Cross Members like Blue Cross Service

Here is a response from one member, and it is typical of hundreds of others on file in the Blue Cross Case Department: "I haven't any suggestions for improvements in the Blue Cro s Plan, but want to tell you what a blessed relief it was to us—just like a light in a storm." It was heartening also to hear Emmett McLoughlin declare at the previously described meeting in St. Monica's that, "Blue Cross Patients are our happiest patients because they have no money worries," and to overhear a hospital admitting clerk tell an incoming Blue Cross patient that she was always glad to see a Blue Cross card because it meant peace of mind for the patient and less work for the hospital office staff.

ASSOCIATED MEDICAL CARE PLANS

A. M. A. HOUSE OF DELEGATES APPROVES SEPARATION OF A.M.A. AND A.M.C.P.

Complete separation of the American Medical Association and Associated Medical Care Plans was approved by the A.M.A. House of Delegates at its 98th Annual Session in Atlantic City, meeting June 6-9, 1949.

Recommended originally by the Council on Medical Service of the A.M.A., in a statement delivered to the Blue Shield Commission of A.M.C.P. at its meeting in Hollywood, Florida, on April 15, 1949, the separation was accepted by the Blue Shield national organiation before the question was placed before the A.M.A. House of Delegates.

E. Vincent Askey, M. D. (California), chairman of the reference committee to which the Council's recommendation was referred, in commenting on the committee's report, said, "Your reference committee feels that it is important that the Delegates read carefully the comment of the Council on Medical Service, appearing in the second paragraph of its recommendation, so there may be no misunderstanding as to the value attached to the accomplishments of A.M.C.P."

The statement referred to in Dr. Askey's word of caution, said, "The Council on Medical Service desires at this time to acknowledge the efforts of A.M.C.P. in promoting through its member plans the principle of voluntary prepayment health insurance; and believes that A.M.C.P. has reached a state of development where it can function more adequately as an autonomous trade association."

In approving another resolution, introduced by L. Howard Schriver, M. D. (Ohio), the House of Delegates pledged its support to A.M.C.P. as an independent federated agency representing state and local Blue Shield Plans.

It was commonly agreed, by all concerned, that one of the reasons for the separation of these two organizations had been an inability to agree upon a Blue Shield proposal to establish a national enrollment agency for handling so-called national accounts. The dilemma was bridged by adoption of the Schriver resolution, which "FURTHER RESOLVED that the several state and local Blue Shield Plans continue the development of an enrollment agency to act in their interest in the field of so-called 'national accounts,' using their best judgment (and that of sponsoring societies) with respect to the methods, means, procedure and form of organization by which the problems related to national accounts may be solved."

Five members of the Blue Shield Commission originally appointed by the Council on Medical Service, were invited by the Commission to continue their membership as individuals, even though they no longer represented the A.M.A. The five Commissioners include Drs. A. W. Adson, Elmer Hess, Charles Gordon Heyd, J. D. McCarthy, and Carl F. Vohs.

Leaders in the Blue Shield movement accepted the change in status as an indication that A.M.C.P. had matured to the point where it could function, move efficiently as an independent trade organization, and without official relationship to the A.M.A. A situation which had become highly controversial was resolved to the apparent satisfaction of everyone involved.

LABOR OFFICIAL ASKS HIGHER INCOME LIMITS FOR SERVICE BENEFITS

Speaking to an assemblage of over four hundred physicians, hospital administrators, business leaders, and friends of Blue Shield and Blue Cross in Kansas City recently, Harry Becker, Director of Society Security for the U.A.W.-C.I.O. plead with the physicians of America to support their Blue Shield Plans and find a way to raise income ceilings on service benefits.

"We urgently and pleadingly ask the physicians throughout America to support their Blue Shield program and to find a way to adjust their program so that persons with incomes below $5,000 will be assured that when illness strikes and hospital care is necessary, the voluntary Blue Shield Plan will meet the whole cost of the doctor's care while the patient is in the hospital," Becker said.

"The average worker does not want cash indemnity, and cash indemnity is a poor substitute for National Health Insurance," Becker continued. "Blue Shield is the only agency that can deliver a full service contract. Commercial insurance cannot do this job. Both Blue Cross

and Blue Shield are in a unique position in that they are tax-free, they are non-profit, and they can make contracts with hospitals and physicians for delivery of service which no insurance company can do.

"What we want in U.A.W.-C.I.O., and what other unions want, is assurance that when our workers go to the hospital, the hospital and doctor bill will be paid. Until we have done that job we have not answered the problem of how we are going to pay for medical care, and we have not provided an alternative to National Health Insurance."

Mr. Becker, in addition to his official responsibility with the largest labor union in America, is a member of the governing boards of both Blue Shield and Blue Cross in Michigan. His appearance in Kansas City was scheduled as the highlight of the annual meetings of Blue Shield and Blue Cross, held jointly at the President Hotel on May 13, 1949.

Telling his listeners that labor recognized the costs involved in providing adequate medical care, Becker indicated that fourth round economic demands for 1949 would be directed toward employer financed health and welfare programs. He expressed, further, the hope that Blue Shield Plans would be in a position to deliver a satisfactory program of prepaid medical care when the chips were down.

Referring to national coverage, Becker stated that he hoped Blue Shield and Blue Cross would "arrange for uniform, national standards of benefits, so that when an agreement is made with an employer for financing hospital and medical costs, the program can be delivered wherever the employee lives, whether the contract was made in Detroit, Pittsburgh, Cleveland, Los Angeles or Kansas City and whether the employees live in one part of the country or another part of the country. We can't do for our members in Michigan what we cannot do for our members in Missouri under the same labor management contract."

Becker mentioned, in passing, the importance of public representation on Blue Shield and Blue Cross Boards of Trustees, basing his whole argument on the great need for all groups, medical, hospital, and labor, to work together toward finding an answer to the people's problem of meeting the costs of health care.

BLUE SHIELD PLANS ENROLLING MORE THAN 10,000 PER DAY

"New members are being enrolled by Blue Shield at a rate of more than 10,000 per day," Frank E. Smith, Director of Associated Medical Care Plans, national coordinating agency for Blue Shield Plans, stated recently.

With a net gain of 966,294 members during the first quarter of 1949, Blue Shield headquarters in Chicago announced recently that enrollment in the non-profit medical prepayment plans had reached a total of 11,333,758 on March 31, 1949.

At the present rate of growth, Blue Shield enrollment passed the 12,000,000 mark during June. First quarter growth represented a gain of 8.82 per cent over the total reported at the end of 1948.

The first quarter gain of 966,294 members was approximately 50 per cent better than the first quarter of 1948, when 645,222 members were added by the Plans.

MORE THAN 100,000 CHECKS ISSUED MONTHLY TO PHYSICIANS BY BLUE SHIELD

More than 100,000 checks are issued each month by Blue Shield Plans, made payable to physicians throughout the United States for services rendered to Blue Shield members.

An estimated 6,000 additional checks are issued by the Plans directly to beneficiary members as reimbursement for expenses incurred each month.

The monthly total of checks written is no indication of the aggregate number of services rendered each month. Some Plans issue cumulative checks, in which a single check is written to cover all services reported during the previous month. Furthermore, only a portion of the checks written each month are in payment for services rendered in the same month, a large volume being issued in payment for services rendered during preceding months, reports on which were not submitted to the Plans during the month in which services were rendered.

VETERANS EXPRESS PREFERENCE FOR HOME TOWN CARE BY OWN PHYSICIANS

California Physicians' Service completed recently a survey among veterans who had been treated by physicians of their own choice under

a program of home town care, administered by the Plan through a contract with the Veterans Administration.

Favorable comments were received from 92 per cent of the thousands of veterans who replied to the San Francisco office of California's Blue Shield Plan.

A majority of 54 per cent reported that free choice of their own physician resulted in the type of personal care which, in their opinion, was better than any other kind of medical attention.

Being treated by their own physicians saved time and trouble, said 15 per cent of the responses, permitting a veteran to continue his employment or education without interruption.

Approximately 10 per cent noted that the program provided relief from congestion in VA hospitals and clinics. The veterans said that they had encountered only a minimum of red tape.

Some said that the program enabled them to maintain their homes, due to relief from financial burdens. Others expressed a desire for continuation of the program.

Only a small minority of 8 per cent were critical of delays in treatment, payment of bills, and regulatory restrictions.

TWO MORE PLANS ACCEPTED AS MEMBERS OF A. M. C. P.

Two more Blue Shield Plans were added to the roster of A.M.C.P. members by the Blue Shield Commission, following its meeting in Atlantic City on June 4, 1949, bringing the total number of members to sixty-four Plans.

A.M.C.P.'s newest members will soon be enrolling subscribers in South Carolina and Tennessee, raising to thirty-eight the number of states being served by Blue Shield Plans.

Blue Shield Plans are also active in the District of Columbia, Hawaii, Puerto Rico, and two Canadian provinces.

NON-MEDICAL BOARD MEMBERS TO BE ADDED BY KANSAS CITY PLAN

Eight non-medical members will be added to the governing board of the Blue Shield Plan in Kansas City, according to an announcement made at the Plan's annual meeting on May 13, 1949.

Kansas City's governing board was limited originally to physicians, the membership now being expanded to permit the adding of prominent business and civic leaders.

"There has been a trend during the last three or four years among Blue Shield Plans to include non-medical members on governing boards," stated Frank E. Smith, Director of Associated Medical Care Plans. "Blue Shield has learned to place a high value on the voluntary services of business and civic leaders as board members. These men contribute immeasurably to the success of the Plan. Furthermore, the presence of outstanding men, outside of the medical profession, on Blue Shield governing boards is entirely logical from a public relations standpoint."

MINNESOTA PLAN RAISING INCOME LIMITS AUGUST 1ST.

With the approval of the Council and the House of Delegates of the Minnesota State Medical Association, Blue Shield in Minnesota will raise the income limitations of subscribers eligible for full service benefits on August 1, 1949.

Single member income limits will be raised from $1500 to $2000 per year, and families with two or more participants will be raised from $2500 to $3000 per year.

During the first three months of 1949 only 20.2 per cent of the total payments to doctors were made for services rendered to persons with incomes below the original limits.

Minnesota's Blue Shield Plan is currently among the most rapidly expanding Plans in the country, having added 51,045 members during its first fifteen months of operation.

HOUSE OF DELEGATES APPROVES BLUE SHIELD PLAN FOR SOUTH CAROLINA

At its annual meeting in Myrtle Beach, S. C., on May 16-18, 1949, the House of Delegates of the South Carolina Medical Association approved the incorporation of South Carolina Medical Care Plan.

A Board of Directors, including eight physicians and seven non-medical members, was elected and given authority to incorporate the Plan immediately. At the first meeting of the Board of Directors in Columbia on May 22, 1949, J. D. Guess, M. D., Greenville, was elected President.

Application for associate membership in Associate Medical Care Plans was authorized and later accepted by the Blue Shield Commission of A.M.C.P. at its Atlantic City meeting on June 4, 1949.

Dr. Guess appointed several committees to complete details pertaining to subscribers' cer-

tificates, participating physician agreements, financing, and general administration, which are to report in September. It is expected that the new Blue Shield Plan in South Carolina will be ready for business before the end of 1949.

Blue Cross will be requested to enter into a joint operating agreement for the administration of the new Plan. $10,000 has been authorized as a capital loan by the state medical society to assist in financing the Plan during its initial period of operation.

PORTABLE AUDIOMETER ANNOUNCED BY SONOTONE

Filling a widespread demand in educational and health fields, Sonotone Corporation of Elmsford, N. Y., leading manufacturer and distributor of hearing aids and other auditory devices, today announced development of a portable, economical screening audiometer of the pure-tone type, the modern device for testing hearing.

Use of an individual audiometer for screening tests of the hearing acuity of school children and other large groups is now recommended by most authorities in place of cumbersome group phonographic equipment or the old-fashioned tuning fork and watch tick tests.

The new Sonotone Model 30 Screening Audiometer, moderately priced, not only provides highly accurate tests of individual hearing but can be easily carried, weighing only 12 pounds complete. Operated on long-life, self-contained batteries, it can be used with complete safety to make rapid examinations under all conditions without dependence on electrical outlets.

The new device was especially designed for use in school hearing conservation programs but on the basis of advance showings to doctors, clinics and educators, it is also expected to prove invaluable to physicians, clinics, hospitals, public health departments, health and personnel departments of industrial organizations, university speech and hearing departments, social workers and welfare and civic associations. It is contained in a rugged case measuring only 17 inches long, six inches wide and seven inches high. Although easy to operate, it provides clinical audiometer accuracy standards.

An audiometer eliminates guesswork in hearing tests by allowing the person being examined to listen to a scientifically produced and precisely regulated series of tone signals covering the frequency range of sounds we commonly hear in everyday life. By indicating to the operator if he is unable to hear some of the signals, the person being tested supplies an appraisal of his hearing capabilities.

In testing school children and other groups with the new audiometer, each person can be given a rapid yet individual screening test which will show whether a hearing loss is evident. Where losses are indicated, the impairments should then be re-examined more thoroughly to determine whether the loss is severe enough to warrant corrective and rehabilitative steps.

The new audiometer was especially designed as a tool in the child hearing conservation program advocated by Sonotone's President, Dr. Irving I. Schachtel, which calls for a hearing test for every school child in the country, at least once a year, for early discovery of hearing losses. If auditory impairments are detected at an early stage in life, Dr. Schachtel points out, remedial steps can be taken which may prevent the tragedy of serious hearing defects.

BOOK REVIEW

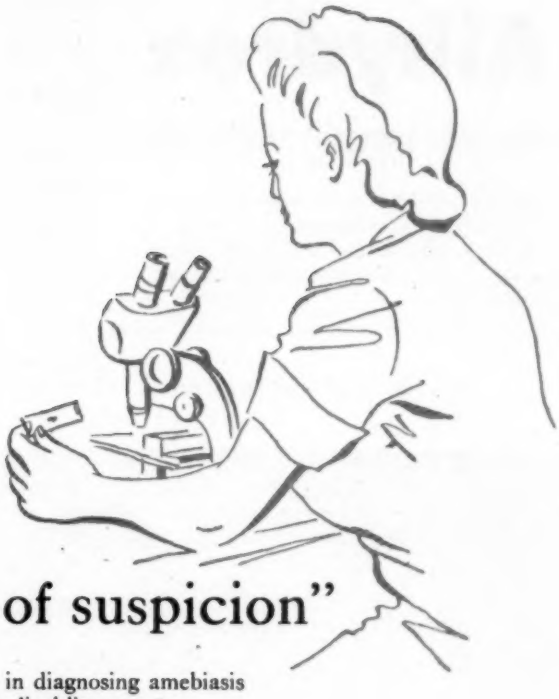
SHEARER'S MANUAL OF HUMAN DISSECTION—2nd Edition. Edited by Charles E. Tobin, Ph.D., Associate Professor of Anatomy, University of Rochester School of Medicine and Dentistry. The Blakiston Company, Philadelphia 5, Pa., Toronto 2, Canada. 79 illustrations; 286 pages; July 13, 1949. $4.50.

This manual or dissecting guide is designed to facilitate and enhance instruction in gross anatomic laboratories. It has achieved a workable balance between the amount of procedure for dissection and descriptive text. Designed to be an autonomous unit, this manual does not have to be used in conjunction with, or with reference to, any specific descriptive text of human anatomy.

It gives the dissection procedure for the entire body and yet keeps the dissected parts in as near their normal relationships as possible, so that relationships as well as individual parts can be studied. The instructor can adapt the plan of this manual to any sequence of regional dissection. Text descriptions have been simplified and illustrations added. New anatomical concepts, developed since the first edition, are included.

CONTENTS: Preface to Second Edition; Preface to First Edition; Introduction.

Pectoral Region; Axilla; Triangles of Neck; Structures under Sternomastoid; Sternoclavicular Articulation; Root of Neck; Back; Head and Neck; Larynx; Thorax; Abdomen; Penis, Scrotum and Testes; Inguinal Region—Inguinal Hernia; Abdominal Cavity; Diaphragm and Posterior Abdominal Wall; Perineum; Pelvis Minor; Superior Extremity; Inferior Extremity. Index.



A high index of suspicion"

The difficulties and pitfalls in diagnosing amebiasis are stressed frequently in medical literature.

"... despite the absence of a history of dysentery, amebiasis must be considered in the differential diagnosis of many bizarre clinical syndromes.... A high index of suspicion is the keynote of early diagnosis."[1]

In acute or latent forms of amebiasis, Diodoquin may be employed over prolonged periods. This high-iodine-containing amebacide "is well tolerated.... The great advantage of this simple treatment is that in the vast majority, it destroys the cysts of *E. histolytica* and is, therefore, especially valuable in sterilizing 'cyst-carriers.' It can readily be taken by ambulant patients and, therefore, eliminates the necessity of hospitalization."[2]

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RESEARCH IN THE SERVICE OF MEDICINE

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1. Warshawsky, H.; Nolan, D. E., and Abramson, W.: Hepatic Complications of Amebiasis, New England J. Med. *235*:678 (Nov. 7) 1946.

2. Manson-Bahr, P.: Some Tropical Diseases in General Practice: "A Post-War Legacy," Glasgow M. J. *27*:123 (May) 1946.

ARIZONA MEDICAL ASSOCIATION MEMBERSHIP ROSTER

Alphabetically Listed

As of July 31st, 1949

PRESIDENTS AND SECRETARIES OF THE COUNTY MEDICAL SOCIETIES

APACHE:
President—A. H. Dysterheft.....McNary
Secretary—E. V. Browning.....Springerville

COCHISE:
President—E. W. Adamson.....Douglas
Secretary—E. E. Royce.....Douglas

COCONINO:
President—M. G. Fronske.....Flagstaff
Secretary—J. H. Calley.....Williams

GILA:
President—A. J. Bosse.....Globe
Secretary—N. D. Brayton.....Miami

GRAHAM:
President—F. W. Butler.....Safford
Secretary—D. E. Nelson.....Safford

GREENLEE:
President—A. E. Engelder.....Morenci
Secretary—Roy E. Burgess.....Clifton

MARICOPA:
President—E. Henry Running.....Phoenix
150 W. McDowell
Secretary—G. G. McKhann.....Phoenix
15 E. Monroe

MOHAVE:
President—F. M. Findlay.....Kingman
Secretary—W. F. Orlando.....Kingman

NAVAJO:
President—M. G. Wright.....Winslow
Secretary—H. B. Potthoff.....Winslow

PIMA:
President—Harry E. Thompson.....Tucson
150 W. McDowell
Secretary—W. B. Steen.....Tucson
110 S. Scott

PINAL:
President—G. H. Walker.....Coolidge
Secretary—Charles R. Law.....Coolidge

SANTA CRUZ:
President—L. B. Milton.....Nogales
Secretary—C. S. Smith.....Nogales

YAVAPAI:
President—John W. Berg.....Clarkdale
Secretary—C. E. Yount.....Prescott

YUMA:
President—J. F. Stanley.....Yuma
Secretary—James Volpe, Jr.....Yuma

Aarni, John C.
Ray

Adams, Mabel I.
301 E. Bethany Home Road
Phoenix

Adamson, Edward W.
Douglas

Alessi, Nicolo V.
Douglas

Allen, Francis W.
18 East 3rd
Tucson

Allen, James H.
Prescott

Anderman, Lorenz
114 W. Adams
Phoenix

Andes, Jerome E.
Wickenburg

Antos, Robert J.
20 W. Riverside Drive
Phoenix

Armbruster, A. Carl
234 N. Central Ave.
Phoenix

Armour, Paul S.
543 E. McDowell
Phoenix

Arnow, Davis I.
Chandler

Atha, Henry G.
1901 W. Campbell Ave.
Tucson

Atonna, Guy B.
Douglas

Atwood, Harry D.
Ajo

Axel, Benjamin J.
Tempe

Baier, Frederic D.
1123 N. 7th St.
Phoenix

Bailey, Perry W.
39 W. Adams
Phoenix

Baker, John S.
2211 N. 16th St.
Phoenix

Baker, Kenneth C.
729 N. 4th Ave.
Tucson

Bakes, Edwin C.
15 East Monroe
Phoenix

Baldwin, Earl R.
110 S. Scott
Tucson

Baldwin, Louis B.
15 E. Monroe
Phoenix

Ballard, Sidney W.
828 N. Tyndall
Tucson

Bank, Joseph
800 N. 1st Ave.
Phoenix

Barfoot, G. Robert
1313 N. 2nd St.
Phoenix

Baritell, Harriet S.
1641 N. Tucson Blvd.
Tucson

Barker, Clyde J.
15 E. Monroe
Phoenix

Barker, Clyde J., Jr.
15 E. Monroe
Phoenix

Barker, Robert D.
15 E. Monroe
Phoenix

Barnes, Broda O.
Kingman

Bassett, George O.
Prescott

Bate, Thomas H.
15 E. Monroe
Phoenix

Bates, William H.
Cottonwood

Bean, Francis J.
Pima County General Hospital
Tucson

Beaton, Lindsay E.
1650 N. Campbell
Tucson

Beck, L. D.
1626 N. Central
Phoenix

Beckwith, Harry S.
Winslow

Bendheim, Otto L.
1515 N. 9th St.
Phoenix

Bennett, John K.
721 N. 4th Ave.
Tucson

Bensema, Clarence E.
1800 E. Speedway
Tucson

Berg, John W.
Clarkdale

Bernfeld, Michael
123 S. Stone
Tucson

Bernstein, Dennis
602 N. 4th Ave.
Tucson

Bewersdorf, Herman L.
208 W. Glenrosa
Phoenix

Bewersdorf, Louise W.
208 W. Glenrosa
Phoenix

Bishop, William A., Jr.
1313 N. 2nd St.
Phoenix

Bishop, William E.
Globe

Blank, Edward
733 W. McDowell
Phoenix

Bloom, Benson
1641 N. Tucson Blvd.
Tucson

Bloomhardt, Samuel I.
15 E. Monroe
Phoenix

Bock, Raymond F.
123 S. Stone
Tucson

Boone, George D.
601 E. 6th St.
Tucson

Borah, Charles E.
15 E. Monroe
Phoenix

Born, Ernest A.
Prescott

Bosse, Alexander J.
Globe

Brady, Thomas A.
650 S. Country Club Dr.
Tucson

Brainard, Hollis H.
2440 E. 6th
Tucson

Bralliar, Floyd B.
Wickenburg

Brayton, Nelson D.
Miami

Brazie, Walter
Kingman

Bregman, Edward H.
521 E. Cheery Lynn
Phoenix

Brewer, William A.
412 W. Roosevelt
Phoenix

Brinkerhoff, David E.
926 E. McDowell
Phoenix

Brooks, Jack E.
312 W. McDowell
Phoenix

Brown, Earl H.
130 S. Scott
Tucson

Brown, Preston T.
1313 N. 2nd St.
Phoenix

Browne, Trevor G.
543 E. McDowell
Phoenix

Browning, Ellis V.
Springerville

Bryant, Ira M.
Mesa

Burgess, Roy E.
Clifton

Butler, Fonzie W.
Safford

Cain, William C.
Yuma

Caldwell, Hayes W.
15 E. Monroe
Phoenix

Calley, John H.
Williams

Campbell, Zeph B.
15 E. Monroe
Phoenix

Caniglia, Sebastian R.
543 E. McDowell
Phoenix

Carlson, Arthur C.
Cottonwood

Carlson, Donald G.
543 E. McDowell
Phoenix

Carrada, Luis N.
175 E. 12th St.
Tucson

Carrell, William D.
123 S. Stone
Tucson

Carreras, Miquel A.
130 S. Scott
Tucson

Case, Paul H.
15 E. Monroe
Phoenix

Causey, Paul S.
926 E. McDowell
Phoenix

Charvoz, Elton R.
1103 E. Culver
Phoenix

Cheiker, Abraham
Tempe

Claypool, Lewis B.
1313 N. 2nd St.
Phoenix

Cleveland, William H.
1313 N. 2nd St.
Phoenix

Clohessy, Timothy T.
15 E. Monroe
Phoenix

Closson, Esther M.
4 E. Congress
Tucson

Clyne, Meade
110 S. Scott
Tucson

Cochran, Hiram D.
110 S. Scott
Tucson

Cocuzzi, Frank A.
Showlow

Cogland, John L.
15 E. Monroe
Phoenix

Cogswell, Howard D.
2440 E. 6th
Tucson

Cohen, Matthew
15 E. Monroe
Phoenix

Cohen, Morris D.
1534 E. Speedway
Tucson

Collopy, Charles E.
Miami

Condon, Daniel J.
15 E. Monroe
Phoenix

Conner, Sharrel K.
926 E. McDowell
Phoenix

Corliss, Philip G.
Somerton

Costin, Max
602 N. 4th Ave.
Tucson

Craig, Carlos C.
1313 N. 2nd St.
Phoenix

Creighton, Carroll C.
Flagstaff

Cron, Cyril M.
Miami

Cruthirds, Archie E.
15 E. Monroe
Phoenix

Cumming, Harry A.
1313 N. 2nd St.
Phoenix

Cummings, Robert H.
15 E. Monroe
Phoenix

Dagres, Lucille M.
543 E. McDowell
Phoenix

Daniell, Charles B.
Eloy

Danto, Julius L.
Holbrook

Davis, Edward W.
Avondale

Davis, James G.
2408 Hermosa Ave.,
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Davis, James W.
33 E. Broadway
Tucson

Davis, Jasper W.
McNary

Davis, W. Claude
33 E. Broadway
Tucson

Day, Merriwether L.
926 E. McDowell
Phoenix

De Pinto, Angus J.
15 E. Monroe
Phoenix

De Young, George M.
521 E. 3rd St.
Tucson

Denninger, Henri S.
Glendale

Dixon, George L.
744 N. Country Club Rd.
Tucson

Donahue, John L.
4 E. Congress
Tucson

Douds, Howard N.
123 S. Stone
Tucson

Duncan, Arnott K.
Douglas

Dysart, Palmer
15 E. Monroe
Phoenix

Dysterheft, Arnold H.
McNary

Eckstein, Albert
1515 N. 9th St.
Phoenix

Edel, Frank W.
738 E. McDowell
Phoenix

Edwards, Bryant B.
521 E. 3rd St.
Tucson

Edwards, Walter V.
Cottonwood

Ehrlich, Joseph C.
1313 N. 2nd St.
Phoenix

Eisenbeiss, John A.
926 E. McDowell
Phoenix

Emert, James T.
1313 N. 2nd St.
Phoenix

Enfield, George S.
15 E. Monroe
Phoenix

Engelder, Arthur E.
Morenci

Engle, David E.
1619 N. Tucson Blvd.
Tucson

Fabric, Clarence C.
120 S. 1st Ave.
Phoenix

Fahlen, Frederick T.
112 N. Central Ave.
Phoenix

Faris, Hervey S.
23 E. Ochoa
Tucson

Farness, Orin J.
721 N. 4th Ave.
Tucson

Felch, Harry J.
15 E. Monroe
Phoenix

Fillmore, Angus J.
Mesa

Findlay, Francis M.
Kingman

Fischer, Oscar G.
Holbrook

Fitzgerald, Guy H.
1746 E. North St.
Tucson

Flinn, Robert S.
15 E. Monroe
Phoenix

Flohr, Martin C.
Tolleson

Flood, Clyde E.
4 E. Congress
Tucson

Forster, Wesley G.
543 E. McDowell
Phoenix

Foster, John
543 E. McDowell
Phoenix

Foster, R. Lee
15 E. Monroe
Phoenix

Fournier, Dudley T.
15 E. Monroe
Phoenix

Franklin, Henry L.
15 E. Monroe
Phoenix

Fraser, George
620 N. Country Club Rd
Tucson

Frazier, V. Eugene
Mesa

French, Harry J.
14 N. Central
Phoenix

Frissell, Ben Pat
15 E. Monroe
Phoenix

Fronske, Martin G.
Flagstaff

Fulk, Murl E.
Glendale

Fuller, Ralph H.
St. Mary's Hospital
Tucson

Furth, William G.
11 W. Jefferson
Phoenix

Fusco, L. Donald
315 E. Dunlap
Phoenix

Gain, Douglas D.
15 E. Monroe
Phoenix

Galison, Louis
Buckeye

Gans, Carl H.
Morenci

Garrison, Isaac L.
540 W. McKinley
Phoenix

Gaskins, Duke R.
15 E. Monroe
Phoenix

Gatterdam, Eugene E.
15 E. Monroe
Phoenix

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Ginn, J. Allen
750 E. McDowell
Phoenix

Gonzalez, Juan S.
Nogales

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112 N. Central Ave.
Phoenix
Goss, Harry L.
316 W. McDowell
Phoenix
Gotthelf, Ed J.
4 E. Congress
Tucson
Graham, Duncan G.
Mesa
Grauman, Samuel J.
403 E. 5th St.
Tucson
Green, John R.
15 E. Monroe
Phoenix
Green, Monroe H.
1137 W. McDowell
Phoenix
Greer, Joseph M.
15 E. Monroe
Phoenix
Gregg, Fred C.
41 E. Jackson
Tucson
Gregg, Harold J.
41 E. Jackson
Tucson
Gregory, T. Richard
3515 N. 12th St.
Phoenix
Griess, Donald F.
721 N. 4th Ave.
Tucson
Gudgal Harry B.
15 E. Monroe
Phoenix
Gunter, Clarence
Globe
Haines, Ronald S.
926 E. McDowell
Phoenix
Haislip, Donald B.
15 E. Monroe
Phoenix
Hall, Kenneth E.
540 E. Dunlap
Phoenix
Hall, Norman D.
1313 N. 2nd St.
Phoenix
Hamer, Jesse D.
15 E. Monroe
Phoenix
Hamer, John D.
Lowell
Hansen, Robert M.
Douglas
Harbridge, Delamere F.
15 E. Monroe
Phoenix
Harker, Glenn L.
Nogales
Harper, Theodore C.
Globe
Harris, Ira E.
Miami
Harris, Karl S.
15 E. Monroe
Phoenix
Hartgraves, Thomas A.
926 E. McDowell
Phoenix
Hartman, George O.
20 East Ochoa
Tucson
Hartman, Stanford F.
926 E. McDowell
Phoenix
Hastings, Robert E.
1811 E. Speedway
Tucson
Hausmann, Richard K.
110 S. Scott
Tucson
Hayden, Edward M.
23 E. Ochoa
Tucson
Hayhurst, Darrell E.
1717 E. Speedway
Tucson
Haynes, Bertram P.
Buckeye
Heim, Delmer J.
110 S. Scott
Tucson
Helm, Hugh M.
Douglas
Henderson, Charles E.
738 E. McDowell
Phoenix
Herbst, Kenneth A.
1502 W. Catalina
Phoenix
Herzberg, Benjamin
1313 N. 2nd St.
Phoenix
Hess, George H.
Bisbee
Hewitt, W. Roy
110 S. Scott
Tucson
Hill, Donald F.
2430 E. 6th St.
Tucson
Hilton, Robert K.
Litchfield Park
Hirsch, Louis
Tucson Medical Center
Tucson
Holbrook, W. Paul
2430 E. 6th St.
Tucson
Holmes, Carl A.
1401 N. 7th Ave.
Phoenix
Holmes, Fred W.
15 E. Monroe
Phoenix
Holton, Stanley W.
Reynolds Metal Co.
Phoenix
Honsik, Frank J.
4308 N. 7th Ave.
Phoenix
Hopkins, Doris F.
15 E. Monroe
Phoenix
Horvath, Charles N.
2930 N. 24th St.
Phoenix
Hough, Henry A.
Prescott
Houle, Emile C.
Nogales
Hoyt, Dorsey R.
15 E. Monroe
Phoenix
Huestis, Charles B.
Hayden
Hughes, Thomas J.
Tempe
Hurianek, Zdenka A.
15 E. Monroe
Phoenix
Hussong, Ruland W.
15 E. Monroe
Phoenix
Irvine, George B.
Tempe
Irwin, Ralph T.
Yuma
Jacobs, Jesse E.
Miami
James, David C.
1103 E. Culver
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James, Howard C.
620 N. Country Club Rd.
Tucson
Jarrett, Paul B.
543 E. McDowell
Phoenix
Jeffery, Millard
517 W. McDowell
Phoenix
Jeffery, Vogel J.
Gila Bend
Jekel, Louis G.
15 E. Monroe
Phoenix
Jennett, Raymond J.
543 E. McDowell
Phoenix
Johnson, James L.
15 E. Monroe
Phoenix
Johnson, Paul A.
25 W. McDowell
Phoenix
Johnson, Phillip L.
928 W. McDowell
Phoenix
Johnson, William A.
Glendale
Jolley, Elvie B.
Jerome
Jones, Robert E.
1313 N. 2nd St.
Phoenix
Jordan, Fred C.
15 E. Monroe
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Joseph, Samuel R.
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1313 N. 2nd St.
Phoenix
Keller, Robert S.
Safford
Kent, Leo J.
2430 E. 6th St.
Tucson
Kent, Melvin L.
Mesa
Ketcherside, Hilary D.
800 N. 1st Ave.
Phoenix
Kilgard, Frank M.
913 N. 3rd St.
Phoenix
Kingsley, Alfred C.
15 E. Monroe
Phoenix
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123 S. Stone
Tucson
Kitt, W. Stanley
4 E. Congress
Tucson
Kittredge, Daniel W., Jr.
Flagstaff
Klein, Jack
2219 S. Sixth Ave.
Tucson
Knight, Frederick W.
Safford
Kober, Leslie R.
15 E. Monroe
Phoenix
Kohl, Harold W.
1811 E. Speedway
Tucson
Kolins, Nathan S.
415 E. 5th St.
Tucson
Kosanke, Harold R.
110 S. Scott St.
Tucson
Kripke, Morton J.
206 E. Main
Mesa
Kroeger, Hilda H.
789 Howard Ave.
New Haven, Conn.
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1313 N. 2nd St.
Phoenix
Kuhlman, Clarence H.
721 N. 4th Ave.
Tucson
Laidlaw, Elizabeth H.
2422 E. Helen
Tucson
Lamb, Harold L.
80 S. Stone
Tucson
Lambrecht, Bert E.
Miami
Laugharn, Charles H.
Clifton
Law, Charles R.
Coolidge
Lawshe, Roger D.
Williams
Lehmberg, Harry B.
Casa Grande
Lemmle, Malwina T.
525 E. Speedway
Tucson
Lentz, Joseph S.
543 E. McDowell
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Leonard, Robert E.
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Lesemann, Frederick J.
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Tucson
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1137 W. McDowell
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Davis Dam, Nevada

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123 S. Stone
Tucson

Lewis, Leo L.
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2720 E. Broadway
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Lightner, Clarence M.
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Tucson

Littlefield, Jesse B.
2432 E. 6th St.
Tucson

ooney, Robert N.
Prescott

Loveless, Phil H.
543 E. McDowell
Phoenix

Lovre, Stanton C.
Morenci

Lutfy, Louis P.
301 W. McDowell
Phoenix

Lyon, William R.
316 E. Speedway
Tucson

Lytton-Smith, James
926 E. McDowell
Phoenix

Mahoney, Dan L.
4 E. Congress
Tucson

Mahoney, Vernon L.
614 N. 4th Ave.
Tucson

Mandel, Meyer M.
909 E. Speedway
Tucson

Manning, Wilkins R.
620 N. Country Club Road
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Manoil, Lazarus
34 W. Lynwood
Phoenix

Marcus, David
3935 E. Pima Ave.
Tucson

Marsh, L. Cody
123 S. Stone
Tucson

Matanovich, Mihajlo
15 E. Monroe
Phoenix

Matts, Robert M.
Yuma

Maxwell, George E.
Coolidge

Mayne, Richard H.
1515 N. 9th St.
Phoenix

McCracken, Paul W.
116 S. 12th Ave.
Phoenix

McFarland, Paul E.
509 W. McDowell
Phoenix

McGilvra, Raymond I.
307 E. Indian School Road
Phoenix

McGovern, Teresa
2516 E .8th St.
Tucson

McGrath, William B.
Route 2, Box 442-A
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McIntyre, Arthur J.
11 W. Jefferson
Phoenix

McKenna, John F.
15 E. Monroe
Phoenix

McKeown, Hilton J.
926 E. McDowell
Phoenix

McKhann, George G.
15 E. Monroe
Phoenix

McNally, Joseph P.
Prescott

McVay, L. Clark
15 E. Monroe
Phoenix

Medigovich, Dushan V.
15 E. Monroe
Phoenix

Melick, Dermont W.
15 E. Monroe
Phoenix

Melton, Bernard L.
15 E. Monroe
Phoenix

Meredith, Daniel T.
234 N. Central Ave.
Phoenix

Merrill, Marriner W.
800 N. 1st Ave.
Phoenix

Meyer, Wallace M.
15 E. Monroe
Phoenix

Mezera, Lad R.
Capitol Annex
Phoenix

Mikell, John S.
1811 E. Speedway
Tucson

Milloy, Frank J.
15 E. Monroe
Phoenix

Mills, C. Selby
926 E. McDowell
Phoenix

Milton, Lee B.
Nogales

Miyauchi, Yukio
Glendale

Moats, Dean Terry
1626 N. Central Ave.
Phoenix

Mock, Delmar R.
Patagonia

Montgomery, Robert E.
Douglas

Moore, James R.
15 E. Monroe
Phoenix

Moore, Robert L.
15 E. Monroe
Phoenix

Mullinex, Merlin E.
Prescott

Mulsow, John E.
3362 N. 46th St.
Milwaukee, Wisconsin

Nagoda, Ed J.
4 E. Congress
Tucson

Neff, Bayard L.
Mesa

Neffson, A. Harry
2510 E. 6th St.
Tucson

Nelson, Donald E.
Safford

Nelson, Willis J.
15 E. Monroe
Phoenix

Neubauer, Darwin W.
401 E. 5th St.
Tucson

Nevins, Charles R.
2258 N. 15th Ave.
Phoenix

Nevins, Roscoe C.
6209 S. Central Ave.
Phoenix

Newcomb, Charles J.
620 N. Country Club Road
Tucson

Noon, Zenas B.
Nogales

O'Brien, Walter M.
Globe

O'Connor, Michael J.
4 E. Congress
Tucson

Ohl, Howard J.
25 W. McDowell
Phoenix

O'Neil, James T.
Casa Grande

Omer, Joy A.
4 E. Congress
Tucson

Orlando, William F.
Kingman

Ovens, James M.
926 E. McDowell
Phoenix

Owens, Lincoln L.
Tombstone

Oyler, Raymond F.
130 S. Scott
Tucson

Palmer, E. Payne
15 E. Monroe
Phoenix

Palmer, E. Payne, Jr.
15 E. Monroe
Phoenix

Palmer, Maxwell R.
521 E. 3rd St.
Tucson

Palmer, Paul V.
15 E. Monroe
Phoenix

Palmer, Ralph F.
15 E. Monroe
Phoenix

Parrish, Frederick W.
Bowie

Pasternack, Bernard
522 N. Tucson Blvd.
Tucson

Patterson, John H.
234 N. Central Ave.
Phoenix

Patterson, William B.
Mesa

Payne, William G.
Tempe

Peterson, Claude H.
Winslow

Peterson, Kenneth E.
7 W. McDowell
Phoenix

Phillips, Eileen A.
522 N. Tucson Blvd.
Tucson

Phillips, Robert T.
150 W. McDowell
Phoenix

Phillips, William A.
Yuma

Piepergerdes, Clarence
Bisbee

Ploussard, Charles N.
15 E. Monroe
Phoenix

Podolsky, Abe I.
Yuma

Pohle, Charles L. von
Chandler

Pohle, Ernest E.
Tempe

Polson, Donald A.
800 N. 1st Ave.
Phoenix

Porter, Dwight H.
800 N. 1st Ave.
Phoenix

Potthoff, Herbert B.
Holbrook

Powell, Charles S.
Yuma

Powell, Melvin J.
Litchfield Park

Present, Arthur J.
23 E. Ochoa
Tucson

Presson, Virgil G.
130 S. Scott
Tucson

Price, Robert A.
2258 N. 15th Ave.
Phoenix

Purcell, George W.
131 S. Scott
Tucson

Purcell, Howard M.
330 W. McDowell
Phoenix

Pyre, Jackman
123 S. Stone
Tucson

Ramenofsky, Abraham
39 W. Adams
Phoenix

Randall, George E.
15 N. 17th Ave.
Phoenix

Randolph, Howell S.
15 E. Monroe
Phoenix

Reed, Wallace A.
926 E. McDowell
Phoenix

Reese, Forrest L.
15 E. Monroe
Phoenix

Reichert, Jacob
301 W. McDowell
Phoenix

Reinoehl, Warren L.
800 N. 1st Ave.
Phoenix

Rhu, Herman S.
130 S. Scott
Tucson

Rice, Alfred G.
Chandler
Rice, Hal W.
Bisbee
Rice, Herbert R.
Flagstaff
Rice, Philip E.
Glendale
Richter, Maurice R.
St. Joseph's Hospital
Phoenix
Ricker, John H.
926 E. McDowell
Phoenix
Rider, Robert E.
Yuma
Risser, Christian F.
338 E. Dunlap
Phoenix
Robb, Mayo
15 E. Monroe
Phoenix
Robbins, Clarence L.
1641 N. Tucson Blvd.
Tucson
Robinson, John S.
Bisbee
Rogers, George K.
926 E. McDowell
Phoenix
Rosenquist, W. Royal
Scottsdale
Rosenthal, Maurice
543 E. McDowell
Phoenix
Ross, Norman A.
15 E. Monroe
Phoenix
Rothman, Samuel I.
1617 E. McDowell
Phoenix
Rowley, Thomas O.
Mesa
Royce, Emery E.
Douglas
Rudolph, Charles W.
12 E. 3rd St.
Tucson
Rudolph, Royal W.
1627 N. Tucson Blvd.
Tucson
Running, E. Henry
150 W. McDowell
Phoenix
Rupp, John J.
2265 N. Norris Ave.
Tucson
Ryerson, Paul M.
1505 E. McDowell
Phoenix
Saba, Joseph,
Bisbee
Sanders, Zack W.
330 W. McDowell
Phoenix
Sanger, Stuart
123 S. Stone
Tucson
Sarlin, Charles N.
123 S. Stone
Tucson
Saylor, Blair W.
130 S. Scott
Tucson
Schell, Donald E.
123 S. Stone
Tucson
Schnabel, Garfield P.
Superior
Schoffman, William F.
926 E. McDowell
Phoenix
Schnur, Leo
Grand Canyon
Schroeder, William F.
926 E. McDowell
Phoenix
Schultz, William M.
110 S. Scott
Tucson
Schutzbank, Friedel B.
4065 E. Cooper
Tucson
Schwartzmann, John R.
433 N. Tucson Blvd.
Tucson
Sechrist, Charles W.
Flagstaff
Secrist, Delbert L.
123 S. Stone
Tucson
Semoff, Milton
522 N. Tucson Blvd.
Tucson
Shembab, Cecilia H.
926 E. McDowell
Phoenix
Shepard, William R.
Prescott
Sherrill, Walter P.
342 W. McDowell
Phoenix
Shetter, George A.
123 S. Stone
Tucson
Shoun, Alexander N.
1800 E. Speedway
Tucson
Shultz, William G.
2448 E. 6th St.
Tucson
Shupe, Reed D.
1103 E. Culver
Phoenix
Sickler, James R.
123 S. Stone
Tucson
Siegal, Henry A.
15 E. Monroe
Phoenix
Singer, Paul L.
39 W. Adams
Phoenix
Smelker, Van A.
4 E. Congress
Tucson
Smith, Charles S.
Nogales
Smith, Leslie B.
926 E. McDowell
Phoenix
Snyder, Bertram L.
15 E. Monroe
Phoenix
Snyder, William
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Soloman, David M.
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Phoenix
Sommerfield, William A.
105 E. Yavapai Rd.
Tucson
Southworth, Harry T.
Prescott
Sprague, Lavern D.
110 S. Scott
Tucson
Stacey, John W.
1613 N. Tucson Blvd.
Tucson
Stanford, Henry J.
614 N. 4th Ave.
Tucson
Stanley, John F.
Yuma
Starns, Charles E.
520 N. Park Ave.
Tucson
Steen, William B.
110 S. Scott
Tucson
Steffens, Henry J. J., Jr.
15 E. Monroe
Phoenix
Stephens, Charles, A. L.
2430 E. 6th St.
Tucson
Stern, Morris E.
1313 N. 2nd St.
Phoenix
Stevens, Robert H.
1313 N. 2nd St.
Phoenix
Stevenson, Arthur C.
750 E. McDowell
Phoenix
Steward, Bernice L.
Coolidge
Steward, Gus B.
Coolidge
Stolfa, Laddie B.
926 E. McDowell
Phoenix
Stolz, Harold F.
614 N. 4th Ave.
Tucson
Storts, Brick P.
1811 E. Speedway
Tucson
Stratton, J. Newton
Safford
Stratton, Robert A.
Yuma
Strauss, James F.
721 N. 4th Ave.
Tucson
Strode, Willard L.
2412 N. Campbell Ave.
Tucson
Stump, Robert M.
3301 W. Van Buren
Phoenix
Sturges, Harold J.
Yuma
Sult, Charles W.
15 E. Monroe
Phoenix
Sult, Charles W., Jr.
15 E. Monroe
Phoenix
Sussman, Marcy L.
800 N. 1st Ave.
Phoenix
Swasey, Lloyd K.
15 E. Monroe
Phoenix
Swenson, Alvin L.
1313 N. 2nd St.
Phoenix
Tappan, Vivian T.
2350 E. Elm
Tucson
Thayer, Kent H.
1313 N. 2nd St.
Phoenix
Thoeny, Oscar W.
1313 N. 2nd St.
Phoenix
Thomas, Charles A.
130 S. Scott
Tucson
Thomas, Naugle K.
130 S. Scott
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Thompson, Alden B.
168 W. Broadway
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Thompson, Harry E.
435 N. Tucson Blvd.
Tucson
Thompson, Hugh C.
110 S. Scott
Tucson
Toland, Virgil A.
509 W. McDowell
Phoenix
Tompkins, Lucian M.
Gilbert
Townsend, Samuel D.
309 E. Congress
Tucson
Truman, George C.
Mesa
Tucker, William P.
Florence
Tuthill, Alexander M.
15 E. Monroe
Phoenix
Tuveson, Leo L.
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Phoenix
Ure, William C.
620 N. Country Club Rd.
Tucson
Urry, Audrey Glen
6540 N. 14th St.
Phoenix
Utzinger, Otto E.
Ray
Van Epps, Charles E.
1313 N. 2nd St.
Phoenix
Van Ravenswaay, Arie C.
2430 E. 6th St.
Tucson
Vernetti, Lucy A.
543 E. McDowell
Phoenix
Veseth, Myron E.
Malto, Montana
Vivian, Charles W.
412 W. Roosevelt
Phoenix
Volpe, James, Jr.
Yuma
Waddell, Emmett P.
1115 E. 6th St.
Tucson
Wagner, Alphonse J.
129 S. Scott
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Walker, Glen H.
Coolidge

Wall, Mark H.
Mesa
Walsh, James S.
Douglas
Walton, Robert E.
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Doctors, kindly notify us of any corrections. They will be rectified in the September issue of the Arizona Medicine Journal.

PERSONAL NOTES

The new administrator of the Tucson Medical Center is **DR. MARTIN F. HEIDGEN** of Elmhurst, Illinois. He succeeds Dr. Clyde Fox who has recently resigned.

Dr. Heidgen is a native of Milwaukee, Wisconsin. He graduated from Marquette and obtained his medical degree from the University of Chicago. He studied hospital administration later at Chicago and Cornell, and his work at Elmhurst for 17 years has been as the superintendent of the DuPage County Memorial Hospital.

Dr. Heidgen is a member of several medical and hospital associations. He is a member of the editorial advisory board of "Hospital Management." He was a major in the medical corps during World War II.

A great many Arizona physicians have been saddened by the death of **DR. MILDRED T. WOOLLEY** in Long Beach, California. Dr. Woolley was the widow of the late Dr. Paul Woolley, a nationally known tuberculosis specialist. She was educated at the Universities of California and Michigan, and had a doctorate in public health.

Dr. Woolley engaged in laboratory research at the Desert Sanatorium in Tucson during the 1930's. In July 1941 Dr. Woolley was appointed acting director of the Arizona State Laboratory, and served until 1944 during the director's absence in the armed services. She has been director of laboratories for the Long Beach Health Department since then.

The Hospital Advisory Survey and Construction Council assists the State Department of Health in the allocation of funds, development of policy, etc. The ex-officio chairman is **DR. J. P. WARD,** and the medical members include **DR. C. B. SALSBURY,** of Ganado; **DR. J. P. McNALLY** of Prescott, and **DR. W. DALE JAMISON** of Phoenix. Miss Jane Rider is director of a similar division of the A.S.D. of H.

The second annual **HEALTH EDUCATION WORKSHOP** was held at Arizona State College at Tempe, June 20-23. **FRANK R. WILLIAMS,** M.S.P.H., Director of the Division of Health Education, A.S.D. of H., was in charge. Among the consultants were **DR. T. H. BUTTERWORTH** of the U.S.P.H.S.; **DR. DONALD DUKELOW** of the A.M.A.; **MR. SIMON McNEELY** of the U.S. Office of Health Education, and **DR. JOHN L. MILLER** of Great Neck, N. Y.

The National Tuberculosis Association has announced the election of **DR. BRODA BARNES** of Kingman, Arizona, as a member of its board of representative directors. **DR. FRED G. HOLMES** of Phoenix has been named as one of the new directors-at-large.

DR. CHARLES C. HEDGES, who joined the State Department of Health in April as director of the preventable disease section, has a notable record in pubilc health work and administration. He has recently been superintendent of the Santa Barbara General Hospital, and previously had held positions as assistant medical director of Johns Hopkins Hospital; superintendent of the Babies' Hospital, Columbia University Presbyterian Medical Center; superintendent Roosevelt Hospital, New York City; field worker for the Rockefeller Foundation and City Health Officer for Savannah, Ga.

DR. HARRY EBBS, assistant professor of Pediatrics and senior physician at the Hospital for Sick Children in Toronto, Canada, gave a clinic of two sessions for the Arizona Pediatric Society.

The article in the May issue of ARIZONA MEDICINE on aseptic technic for tuberculosis has been selected for abstracting and national distribution in November by the National Tuberculosis Association. The author, **DR. W. H. OATWAY, JR.,** of Tucson, is on leave at Barlow Sanatorium in Los Angeles.

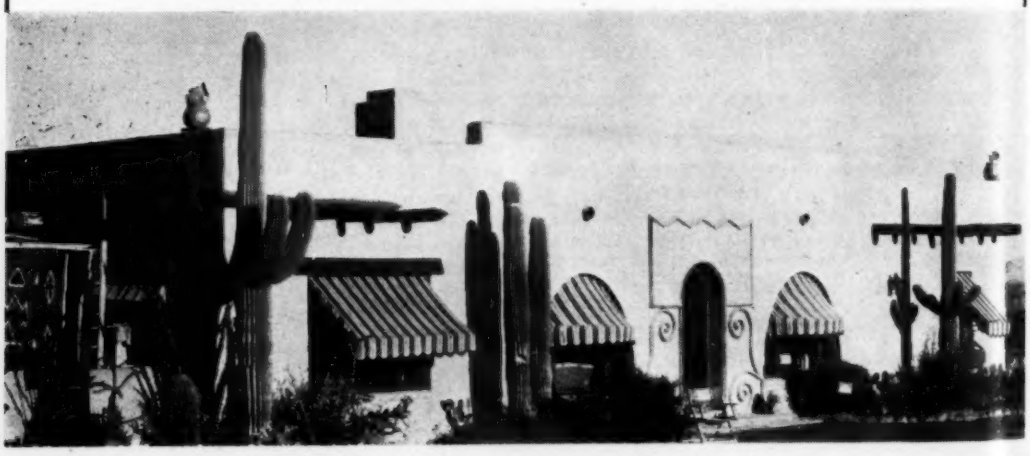
The **SALT RIVER BLOOD BANK** has been established in a similar manner to the Southern Arizona Regional program. The American Red Cross is the sponsor. The two banks have made a reciprocal agreement for help in case of emergency in either area.

Two physicians from San Diego County, California, have retired from practice and moved to the Tucson area. **DRS. ROBERT E. MERRITT** and **L. W. MANSUR** have purchased a ranch on the Bear Canyon road.

A cross-sectional sanitation survey in Maricopa county has demonstrated a notable change during the past two years. The food and drink establishments now have a 67 per cent rating as compared to 48 per cent in March 1947. The surveys are made by the state health department.

DRS. LEWIS HOWARD, EDWARD BRUENING, and **ELIZABETH LAIDLAW** discussed the proposed plans for a health program and revolving health fund for the Pima County and Tucson city schools at a recent school meeting in University Heights.

A severe outbreak of intestinal diseases occurred in Pinal and Maricopa counties in June. The cases included dysentery and paratyphoid infections, and were blamed on contaminated food and polluted water. Most of them occurred in areas with poor sanitary facilities, and the investigation was led by **DR. H. GILBERT CRE-**



CELIUS, director of the state laboratories. There were many cases among the San Carlos Indians including numerous infants; **DR. SANDOR SZITTYA** was the federal medical officer in charge on the reservation. The death of many infants in Phoenix made the outbreak the worst in years.

A new 55-bed hospital is nearing completion in the Arizona State Prison in Florence. It is being built by the prisoners, and is chiefly for care of the tuberculous.

More than 300,000 case-finding x-rays had been taken by June 1st, 1949 since the onset of the state program in May 1945 by the Department of Health.

The board of control of the **ARIZONA STATE HOSPITAL FOR THE INSANE** has ordered several vital medical personnel increases and business office changes. A full-time dentist, as required by law, is to be employed. The staff is to be increased from six to eleven physicians. A staff of surgeons is to be established, using the funds which have been voted for "advanced curative treatment.' An out-patient social service officer is to be appointed to maintain contact with applicant and discharged patients. An office manager with C. P. A. training is to be obtained.

Election to membership in the American Radium Society at its annual meeting in Atlantic City June 1949, has been bestowed on **R. LEE FOSTER,** Phoenix.

A three-year residency in dermatology has been started by **DR. WM. SNYDER,** Phoenix. The study is at the Cincinnati General Hospital, Cincinnati, Ohio.

The following Arizona physicians attended the Ninety-eighth Annual Session of the American Medical Association in Atlantic City June 7-10, 1949:

PHOENIX:—F. D. Baier, Joseph Bank, S. R. Caniglia, Robert S. Flinn, R. Lee Foster, Jesse Hamer, R. I. McGilvra, Wm. Snyder, Robert H. Stevens, C. E. VanEpps and J. P. Ward.

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SOMERTON:—P G. Corliss.

MESA:—Melvin L. Kent.

WHIPPLE:—Morris Rubenstein.

YUMA:—Ralph T. Irwin, James Volpe, Jr.

DR. JESSE HAMER, Phoenix, was delegate from the Arizona Medical Association to the A.M.A. House of Delegates.

DR. D. F. HILL and **W. P. HOLBROOK,** Tuc-

son, presented a paper on "Deformities in Rheumatoid Arthritis" at the A.M.A. Convention, before the Internal Medicine and Experimental Medicine and Therapeutics section.

$150,000 MODERNIZATION OF PHOENIX, ARIZONA, HOSPITAL NEARLY FINISHED

Complete modernization and air conditioning of the west wing of the Good Samaritan Hospital, 1033 East McDowell Street, Phoenix, Arizona, is virtually complete, it was announced today by G. M. Hanner, Administrator of the institution.

Included in the $150,000 modernization project is the installation of the first hospital elevator of its type in the state of Arizona. The new elevator, with a rated capacity of 26 passengers, is large enough to accommodate a streteher, bed or iron lung with an attendant. It is being installed by the Otis Elevator Company at a cost in excess of $20,000 to replace a small, old-style elevator that had been in operation since the hospital was built in 1923. It will rise 35 feet and doors will open automatically as the car levels itself at each stop. Equipped with Collective Control, the elevator will answer only "up" calls as it rises and "down" calls will remain registered until answered on the downward trip.

The four-story-and-basement brick hospital contains 220 beds and is now completely modern and fireproof, according to J. O. Sexon, president of the board. The original building was erected in 1923, a wing was added in 1931 and a nurses' home built since the war. In addition to the new special hospital type elevator, the building is served by two smaller Otis automatic passenger elevators.

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American Cancer Society, Inc.

FROM: Brewster S. Miller, M. D., Assistant Director, Professional Educational Section, American Cancer Society.

SUBJECT: British American Exchange Fellowships in Cancer Research, announcement of.

1. During a visit of the delegation from the British Empire Cancer Campaign to the National Office of the Society last summer, an international exchange of fellowship in cancer research was discussed with The Right Honorable Lord Horder, Vice-Chairman of the Campaign, and his colleagues.

2. Since that time an exchange fellowship program has evolved whereby a number of American investigators in fundamental cancer research and clinical investigation in cancer will study on a fellowship basis in Great Britain where opportunities exist for study in facets of research in malignant disease not widely available here. Provisions have been made for training an equal number of young British scientists selected by the Campaign at research centers in this country.

3. The Committee on Growth has graciously consented to screen applicants desiring to study in Great Britain under this program and will recommend to the Society their selection of candidates on the basis of an application similar to those fellowships granted by the Committee on Growth.

4. Attached find a leaflet outlining the regulations governing these fellowships. We would appreciate your making this program as widely known as possible throughout your Division by means of your state medical society journal, local announcements and publications from the Division, and other means of publicity which you deem to be most effective for your Division.

5. Please note that application forms for these fellowships may be procured from the Executive Secretary of the Committee on Growth and these application forms should be submitted directly to him.

NAVY DEPARTMENT
Office of Naval Officer Procurement
Room 503, 626 South Spring St.
Los Angeles 14, California

Word has been received from the Navy Department by Lieutenant Commander William B. Garrison, U. S. Navy, Director of Naval Officer Procurement, with offices at 626 S. Spring St., Los Angeles, that fifteen hundred (1,500) Naval Aviation Cadets will be appointed during the Navy fiscal year beginning July 1st. These men must be between the ages of 18 and 27, must have completed at least two full academic years toward a degree at an accredited college, university, or junior college, be unmarried and meet the physical requirements for Naval Aviation which includes normal color perception, hearing, pulse and 20-20 vision.

Naval Aviation Cadets receive $75.00 per month, board, lodging, required uniforms and the premiums on $10,000.00 worth of National

Service Life Insurance is paid by the government. They are sent to aviation school for eighteen months at the "Annapolis of the Air" at Pensacola, Florida, and then commissioned as Ensigns in the U. S. Naval Reserve or Second Lieutenants in the Marine Corps Reserve with pay at $318.00 per month without dependents, $399.00 with dependents.

Naval Aviation Cadets become eligible for transfer to the Regular Navy or Marine Corps after completion of eighteen months of commissioned service in such numbers as may be required to meet the needs of the Service.

THE CENTRAL ASSOCIATION OF OBSTETRICIANS AND GYNECOLOGISTS

The eighteenth Annual Meeting of the Central Association of Obstetricians and Gynecologists will be held Thursday, Friday, and Saturday, September 21, 22 and 23, 1950 at the Hotel Schroeder, Milwaukee, Wisconsin.

THE SOUTHWEST SURGICAL CONGRESS

The Southwestern Surgical Congress meeting will be held September 26th, 27th and 28th, 1949, at the Shamrock Hotel in Houston, Texas. All Arizona doctors are invited to attend.

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*Cecil, R. A.: A Textbook of Medicine. Philadelphia, W. B. Saunders Co., 1947, p. 376.

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